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VOLUME 10 NUMBER 5

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ARCHIVES OF OTOLARYNGOLOGY

VOLUME 10 NOVEMBER, 1929 NUMBER 5

THE RELATION OF THE AORTA TO ESOPHAGOSCOPY *

J. SANFORD MOYER, M.D.

PHILADELPHIA

The intimate relation of the aorta to the esophagus renders it a structure always to be taken account of in esophagoscopy,[1] yet in practice the healthy aorta causes the endoscopist little concern. The normal aorta offers no resistance to the passage of the esophagoscope, and when during esophagoscopy the position of the patient is properly maintained, there is little danger of inflicting injury on the aorta.

The arch of the aorta, in its course posteriorly to reach the vertebral column, crosses to the left side of the esophagus. The aorta in its first relationship, therefore, lies anterior and to the left of the esophagus. At a more inferior level, in the lower thoracic region, the aorta passes behind the esophagus, and at the level of the diaphragm it lies posterior and slightly to the right.[2] In its course backward, the arch of the aorta normally impinges on the esophagus and produces a slight constriction of the esophageal lumen at the point of impingement. This occurs at about 23 cm., measuring from the upper incisors.[3] The constriction is entirely physiologic and does not interfere with the function of swallowing. There is no dilatation of the esophagus above the aortic level, and fluoroscopically, one sees that the opaque mixture encounters no obstruction here but passes readily downward.

The Jackson progressive high-low positions of esophagoscopy [4] tend to keep the advancing tip of the esophagoscope away from contact with the aorta; i. e., the head of the patient is at first maintained in a high position. This directs the tip of the esophagoscope back against the

* Submitted for publication, June 12, 1929.

* From the Chevalier Jackson Bronchoscopic Clinics, Graduate School of Medicine, University of Pennsylvania.

* Read before the Section on Otology and Laryngology of the College of Physicians of Philadelphia, Jan. 16, 1929.

1. Christopherson, J. B.: Lancet **1**:369 (March 10) 1917.

2. Spalteholz, W.: Hand Atlas of Human Anatomy, ed. 3, Philadelphia, J. B. Lippincott Company, 1922. Eycleshymer, A. C., and Shoemaker, D. M.: Cross Section Anatomy, New York, D. Appleton & Company, 1911.

3. Jackson, Chevalier: Bronchoscopy and Esophagoscopy, ed. 2, Philadelphia, W. B. Saunders Company, 1927, p. 68.

4. Jackson, Chevalier: Peroral Endoscopy and Laryngeal Surgery, St. Louis, Laryngoscope Co., 1914, p. 78.

spine and away from the aorta, which in its first relationship is anterior to the esophagus. Then, as the tube is passed farther down into the mid-thoracic esophagus and farther, the head of the patient is lowered progressively, thus raising the distal end of the esophagoscope, directing it anteriorly, and protecting the aorta which now occupies a position posterior to the esophagus. In the usual course of esophagoscopy the normal aorta, although lying closely approximated to the esophagus in its lower two-thirds, is not a source of serious risk and causes no great concern.

In disease, however, the aorta presents a serious problem in many cases. The contingency of a dilated aorta must always be borne in mind before every esophagoscopy,[5] and sufficient physical and roentgen examinations made to rule it out. When present, aortic disease with structural impairment must be regarded as a relative contraindication to esophagoscopy, but not an absolute contraindication. For example, when a case of esophageal foreign body is complicated by the presence of a definite aortic aneurysm, it is justifiable to perform an esophagoscopy for removal of the foreign body, although this may be postponed if the patient is able to swallow liquids.[6] For diagnosis in cases in which aortic aneurysm is suspected, esophagoscopy may be difficult and the advisability of attempting it must be determined in the individual case; but with a tube of the proper size a careful esophagoscopy can frequently be done in the presence of an aneurysm with no ill effects. Such an instance was the first case reported below, in which an esophagoscopy was done to determine the diagnosis and evidence of aneurysm secured. Subsequently, multiple aneurysms of the aorta were demonstrated.

The results of aortic pressure on the esophagus are well known: compression of the esophageal lumen, stenosis with dysphagia, esophagitis and ulceration.[7] Varying degrees of stenosis of the esophagus are not uncommonly associated with aortic aneurysm.[8] The pathologic condition is clear, the mechanics of the situation easily understood and the esophageal symptoms readily explained.

Recently, three patients with compression stenosis of the esophagus due to aortic disease were seen in the clinic, and in each the condition was atypical. It was the atypical features of these cases that made them appear sufficiently interesting to report. The first case was one of

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5. Osler, William, and McCrae, Thomas: Principles and Practice of Medicine, ed. 10, New York, D. Appleton & Company, 1925.

6. Jackson, Chevalier (footnote 3, p. 319).

7. Thompson, St. Clair: Diseases of the Nose and Throat, ed. 3, New York, D. Appleton & Company, 1926, p. 641.

8. Ritchie and Ballantyne: Diseases of the Heart, London, E. Arnold & Company, 1922, p. 570. Osler: System of Medicine, ed. 2, New York, Lea & Febiger, 1915. Hirschfelder: Diseases of the Heart and Aorta, ed. 3, Philadelphia, J. B. Lippincott Company, 1919, p. 641.

stenosis due to multiple aneurysms of the aorta; the second was a case of compression of the esophagus due to aortic encroachment not associated with aneurysm, and the third was a case in which aortic compression played a part, but not the chief part in producing esophageal symptoms.

REPORT OF CASES

CASE 1.—A woman, aged 62, suffered from difficulty in swallowing and choking sensations in the throat when taking food. The condition had been present for about three months. At the onset she was able to eat most solid foods, cereals, etc., but gradually she was forced to give up solid foods, as they were regurgitated. Regurgitation occurred soon after meals, within from five to ten minutes. She had lost 40 pounds (18.1 Kg.) during the preceding three months. She also

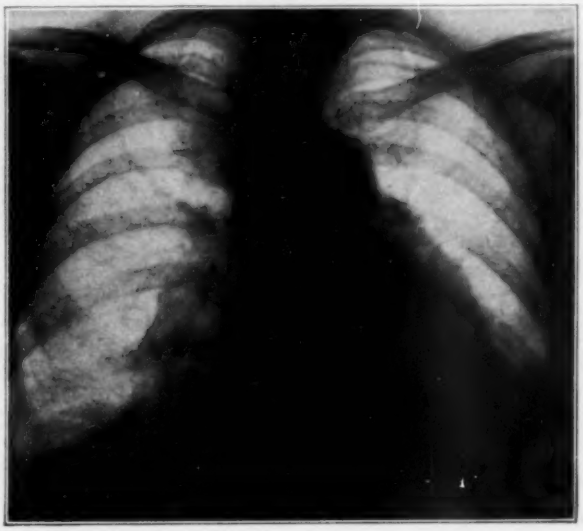

Fig. 1 (case 1).—Roentgenogram which apparently shows a tumor of the mediastinum. This film of the chest, made with the usual technic to bring out lung structure, failed to disclose the important pathologic conditions present. The cardiothoracic ratio is increased. The arch of the aorta is displaced slightly to the left, but does not appear in this film to be aneurysmal. This film should be compared with figure 2. Roentgenographic studies and films of the patients here reported were made by Dr. George E. Pfahler and Dr. J. Gershon Cohen.

gave a history of having been somewhat dyspneic for a period of ten years; the dyspnea had been rather severe during the past three years. Recently, she had had several distressing attacks of dyspnea associated with a sense of pressure and pain in the chest, edema of the ankles and other symptoms of cardiac decompensation.

On admission to the hospital, the most important observations on physical examination were in the chest and abdomen. The chest was narrow and the sternum prominent. The whole precordium heaved with each cardiac pulsation. The heart was considerably enlarged, chiefly to the left. A systolic murmur heard at the apex was transmitted to the axilla. The aortic second sound was accentu-

ated and snapping, and the aortic arch was widened. The pulse pressure in the left arm was 20 mm. less than in the right. There were no other vascular phenomena peculiar to aortic aneurysm, and no pupillary, laryngeal or significant tracheal signs were observed. In the abdomen a mass about the size of a lemon was palpable in the epigastric region, having a definitely expansile pulsation. The Kolmer-Wassermann and Kahn tests were negative.

Because of the esophageal obstruction, a diagnostic esophagoscopy was deemed advisable. The possibility of the obstruction being due to pressure on the esopha-

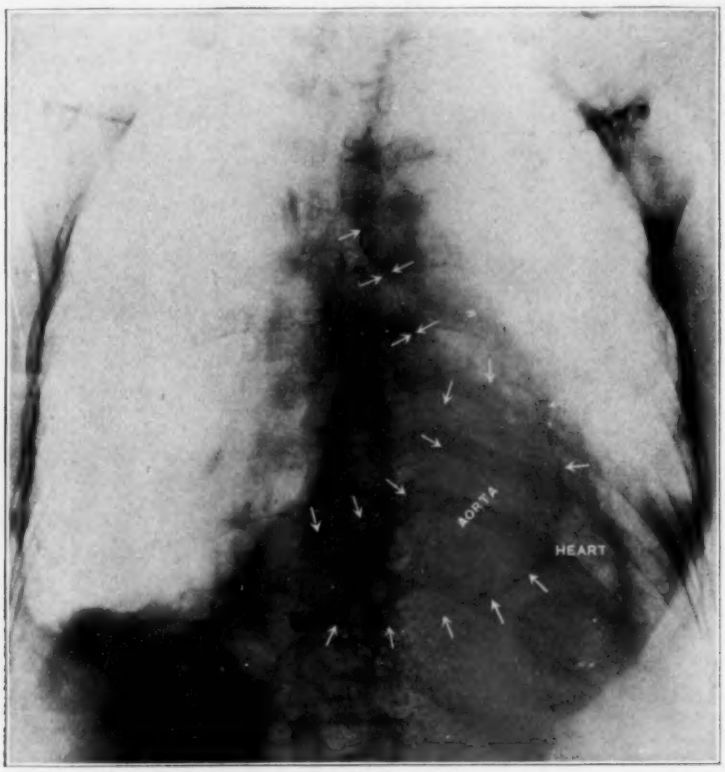

Fig. 2 (case 1).—Roentgenogram, taken in exactly the same position as that shown in figure 1, but with high penetration technic to bring out structures within the cardiac silhouette. What appears to be a mediastinal tumor in figure 1 is shown to be an extremely tortuous aorta with marked calcification of its walls. It should be noted that the aorta, indicated by arrows, occupies a horizontal plane in the lower thorax. Compare with figure 3.

gus by the aorta was kept in mind, and the greatest care was employed in making the examination. The thoracic esophagus was found to be widely dilated, with considerable retention of secretion. Just above the level of the diaphragm, the posterior esophageal wall was pushed forward by a firm mass which showed pulsation, not violent, however. The mass, intruding on the posterior esophageal wall, produced a compression stenosis at that level, through which it was not thought advisable to pass the esophagoscope farther.

The result of the roentgen examination, made by Dr. George E. Pfahler and Dr. J. Gershon Cohen, was interesting. In figure 1 is shown the anteroposterior view of the chest, made with the usual technic for showing lung detail. In this film one sees what appears to be a mass in the mediastinum. The aorta is somewhat widened, but from this view one would scarcely call it aneurysmal. The trachea is displaced to the right. There is an evidence of cardiovascular disease in the hypertrophy of the left ventricle, the cardiothoracic ratio being considerably greater than normal. This film is included chiefly to demonstrate that a great

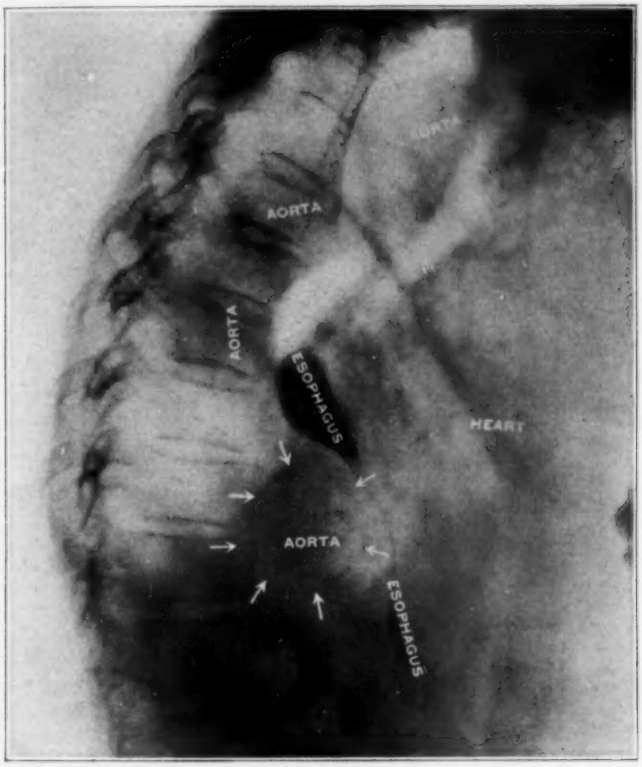

Fig. 3 (case 1).—Opaque material in the lower esophagus outlines a pressure stenosis produced by a rounded mass (aorta). The aorta, indicated by arrows, is seen bulging into the esophagus from behind. Figure 2 explains why the aorta is seen here, in the lower thorax, in cross-section.

deal of pathologic change of interest to the esophagoscopist, pathologic change concerning the lower esophagus, can be entirely missed in the ordinary film.

The film shown in figure 2 was taken in exactly the same position as the preceding one, but the density of the heart shadow has been pierced by the use of the Bucky diaphragm and a special penetration technic. A rounded mass outlined by peripheral deposits of calcareous material is visible. This appearance favored the diagnosis of a large tumor in the mediastinum with calcareous degeneration, or some large cystic formation, possibly a dermoid or an echinococcus cyst. The question of mediastinal tumor created one of the most difficult diag-

nostic problems of this case, and esophagoscopy aided in solving this problem. If in this case the esophageal obstruction was due to a mediastinal tumor compressing the esophagus, one would consider it more probable that the pressure would be exerted from an anterior direction. But esophagoscopy showed the compression to be entirely from the posterior direction. This fact favored the aorta as the cause of the compression, rather than a mediastinal tumor. When this film is studied with reference to other views in this case, it is seen that the mass is not a tumor of the mediastinum, but an atypical aorta, and the calcareous deposits are plaques in the walls of the aorta. The aorta is aneurysmal and extremely tortuous, so tortuous, in fact, that behind the heart, just before it pierces the diaphragm, the aorta does not run in a vertical direction, but occupies a horizontal plane; it takes a course along the upper surface of the diaphragm, from left to right.

Fig. 4 (case 1).—Compression stenosis of lower esophagus, as shown also in figure 3. Note that the arch of the aorta is elongated and undulating in outline.

The lateral view of the chest, figure 3, shows how the esophagus is compressed. The mass that is causing the esophagus to be pushed forward contains a distinct, well formed, peripheral ring of calcification. Normally, in this location, in a lateral view of the chest, the aorta is seen as a vertical, elongated shadow, not as a circle (fig. 3). When figure 3 alone is studied, without reference to other roentgenographic views of this chest, it is hard to conceive how this circular shadow can represent the aorta. But referring to figure 2, it is noted that in this case the aorta is extremely tortuous and that a lateral view of the chest would give a cross-sectional or end-on view of the aorta in the lower thorax. The aorta, therefore, appears as a circular shadow, and the calcareous deposits in its walls form a fairly distinct ring. This case demonstrates the necessity of having an anteroposterior film taken with high penetration technic in order to bring out the details of the aorta within the cardiac silhouette. Without the anteroposterior film (fig. 2), it would be hard to interpret the calcareous ring seen behind the esophagus in the lateral view (fig. 3). The anteroposterior film (fig. 1), taken

with the ordinary technic used in photographing details of the lungs, did not give any indication of the actual condition of the aorta.

The film shown in figure 4 was taken in the same position as that in figure 3, but without the use of high penetration technic. The marked compression stenosis of the lower part of the esophagus is demonstrated.

While in the hospital this patient was treated for a time by antisyphilitic measures, but this treatment gave little relief. About two months after admission to the hospital, death occurred by spontaneous rupture of one of the aneurysms into the left pleural cavity. At autopsy, multiple aneurysms of the

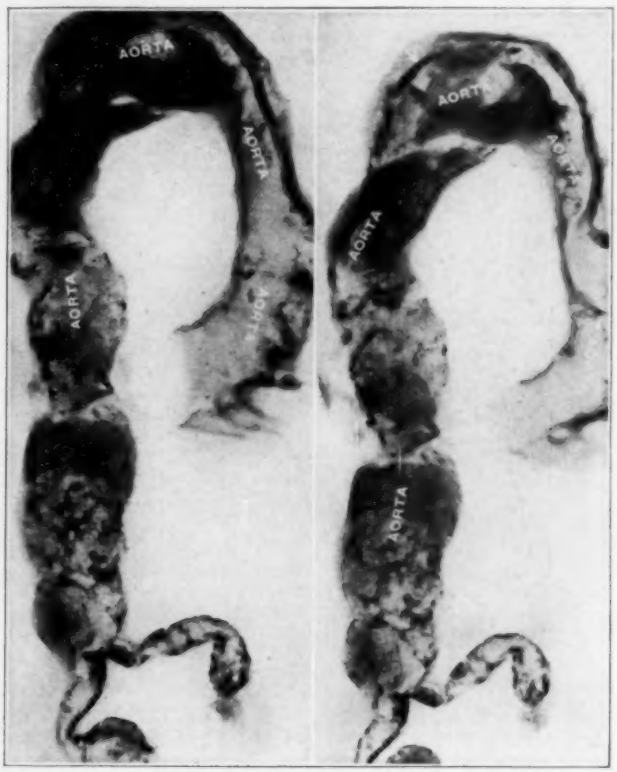

Fig. 5 (case 1).—Roentgenogram of the aorta after it was removed at autopsy. The extreme tortuosity, the multiple aneurysms and the extensive calcification should be noted. This verifies the unusual roentgen manifestations in this case, as shown in the preceding illustrations.

aorta were found. In this case, the etiologic factor in the development of the aneurysms was an arteriosclerotic process. Evidences of syphilis were not found.

A roentgenogram was made of the aorta of this patient after it had been removed at autopsy. The film, shown in figure 5, illustrates the extreme tortuosity, the calcareous degeneration and the multiple aneurysms which produced the unusual roentgenographic and endoscopic picture observed during life.

Case 2.—Mrs. E. E., aged 57, over a period of several years had had occasional choking attacks, especially when eating solid food. Since August, 1928 (three months previously), the condition had been definitely more marked. She had a sensation of food collecting high up in the throat and causing choking; to gain relief, she was forced to induce regurgitation by insertion of the fingers. There was no actual pain on swallowing. Physical examination of this patient showed nothing referable to an esophageal lesion. There was no adenopathy. No increase in aortic dulness was demonstrated, but there was a systolic murmur at the apex, not well transmitted. Other physical observations were unimportant. Because of the dysphagia an esophagoscopy was done, and the esophageal lumen was found to be narrowed at the level of the aortic arch. The anterior wall seemed to be bulged backward, and there was a distinct pulsation at the point of

Fig. 6 (case 2).—Film taken in the oblique position. The opaque material outlines the compression stenosis of the esophagus produced by the arch of the aorta. Note the incisura and niching just below the aortic level.

compression. The mucosa of the esophagus showed a superficial ulceration, but no fungations. Complete exploration of the esophagus revealed nothing otherwise abnormal.

In figure 6 is seen a roentgenogram of this patient, taken in the oblique position. In this film the opaque mixture outlines a bulging which the aortic arch produces into the esophagus. The pathologic change in the aorta in this case appears to be an elongation with atherosclerosis, but not aneurysm. However, the opaque material does not flow over the obstruction produced by the aorta as smoothly as it would if the obstruction were the only pathologic condition present. The incisura and sharp angular niching, seen just below the aortic compression, indicate that there is also some ulceration associated with periesophageal adhesions; as already noted, this suspicion was confirmed by endoscopy. That a tortuous, atherosclerotic aorta can produce symptoms similar to the symptoms of aneurysm was dem-

onstrated long ago.[9] In 1903, Sailer and Pfahler reported a series of cases indicating that tortuosity of the aorta, due to arteriosclerosis, may strongly suggest the presence of aneurysm. In this excellent report eighteen cases were recorded in which the vascular, laryngeal and tracheobronchial signs and symptoms commonly associated with aneurysms of the arch of the aorta were observed in patients who in most instances were definitely free from aneurysms but affected with aortic disease involving sclerosis, elongation and tortuosity. None of these patients had esophageal symptoms, however. An elongated, sinuous aorta may produce pressure effects on surrounding structures. Doubtless this is the basis of the esophageal compression encountered in the present patient.

Case 3.—In Mrs. L. W., aged 70, esophageal symptoms dated back four months, with occasional choking while eating. At the onset, when the choking occurred she was able either to swallow the food or to bring it up again. The condition became steadily worse. A short time prior to admission to the hospital,

Fig. 7 (case 2).—Another roentgenogram showing only a small amount of the opaque meal remaining in the esophagus at the stenosed level. The arch of the aorta encroaches on the esophageal lumen to such an extent that it appears only as a thin crescent lying over the convexity of the aortic wall. The aortic arch is elongated and sclerotic.

while eating lunch she choked on a morsel of meat and was not able to dislodge it. Immediately thereafter she was unable to swallow anything; even saliva was regurgitated. Esophagoscopy was done by Dr. Jackson, and the bolus of meat was removed. She was then able to swallow liquids and soft food. This patient had cancer of the breast which was treated six years before by radium, and since then she received roentgen treatment.

The physical examination in this case was interesting. There was a firm, hard mass in the supraclavicular fossa of the right side. This was on the same side as the cancer of the breast and appeared to be a metastasis from it. But on the left side, there was unilateral recurrent paralysis of the larynx. This raised

9. Sailer, J., and Pfahler, G. E.: Am. J. M. Sc. **126**:616, 1903.

the question of whether the left recurrent nerve was involved by smaller metastases, not visible or palpable at present, or whether the left recurrent nerve was affected by the aorta; the latter seemed more likely. The pathology of this condition is given below. The thyroid was not palpable. The heart was apparently normal. The serologic tests were negative.

An instructive roentgenogram of this patient is seen in figure 8. The aortic arch is elongated and atherosclerotic and produces a compression stenosis of the esophagus at *A*. The compression stenosis, as outlined by the opaque meal, begins at the level of the aortic arch *A*, but just beneath this level there is another constriction, *B*, which is even more marked than the upper one produced by the aortic arch. The pathologic process producing this lower constriction is not definitely demonstrated in this film, but it is evident that there are two con-

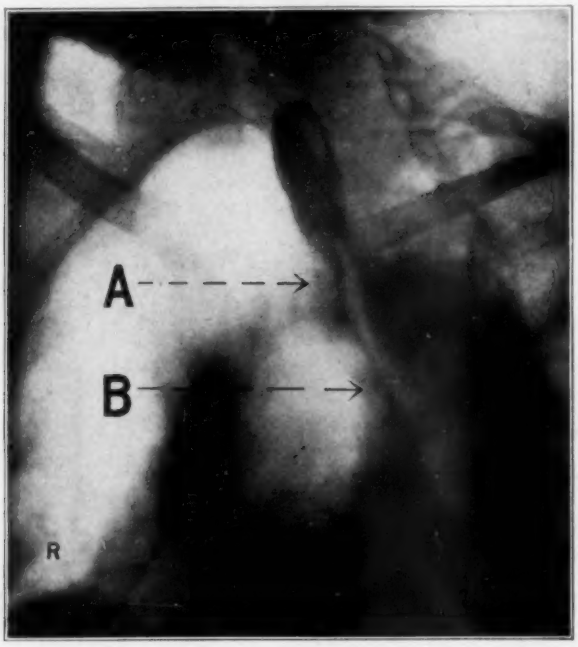

Fig. 8 (case 3).—Stenosis of the esophagus due to compression by the arch of the aorta is demonstrated at *A*. Below this, there is another more marked constriction at *B*, due probably to a periesophageal malignant growth.

strictions of the esophagus: the upper at the aortic level, and the lower, the more marked stenosis, slightly below it. This corresponds to the condition found by esophagoscopy. Figure 9 is a film of this patient taken in the oblique position and in the absence of an opaque medium in the esophagus. The aortic arch is much elongated, atheromatous and tortuous, but not aneurysmal. Compression of the trachea by the arch of the aorta is evident, and it is interesting to note with reference to this tracheal condition that the patient complained of expectoration of mucus and occasional "bronchial colds" dating back to approximately the time of the onset of the esophageal symptoms.

A diagnostic esophagoscopy was done on this patient subsequent to the removal of the foreign body, and the endoscopic observations confirm the results of the

roentgen examination. The arch of the aorta was found to intrude on the left wall of the esophagus at the normal location more than normally. Passing downward a short distance below the aortic level, a firm, infiltrative stenosis was encountered. The lumen through this stenosis appeared to be about 5 mm. in diameter. The esophageal folds on the right side were edematous; on the left wall, the mucosa was inflamed and free from folds. The diagnosis made by esophagoscopy was organic stenosis of the esophagus, probably due to a periesophageal malignant growth located slightly beneath the level of the constriction caused by the aortic arch.

To make a final diagnosis of the nature of the periesophageal growth, it will be advisable to reexamine the patient esophagoscopically at the end of about two months. In the meantime it will be necessary to watch the diet carefully to see

Fig. 9 (case 3).—Film of the chest taken in the oblique position. Note the elongation, tortuosity and sclerosis of the aortic arch, in which atheromatous plaques can be seen. There is also evident compression of the trachea. See text.

that no food is taken that will not pass the constricted area. If any food does lodge again, it will be necessary to remove it esophagoscopically as soon as possible to prevent the development of esophagitis.

The much to be condemned method of removing foreign bodies from the esophagus by pushing downward blindly with a bougie is particularly dangerous when the esophageal stenosis causing lodgment of the foreign body is due to pressure on the esophagus by the aorta.[10] Although not

10. Lovett, J.: Brit. M. J. **1**:1064 (May 1) 1909.

entirely free from risk, esophagoscopy for removal of foreign bodies from the esophagus in the presence of aortic disease is unquestionably the safest method.

CONCLUSIONS

1. The Jackson progressive high-low positions tend to protect the aorta in esophagoscopy.

2. Aneurysm of the aorta is not an absolute contraindication to esophagoscopy for diagnosis.

3. Without special roentgen technic, a pathologic condition in the lower part of the thorax of interest to the endoscopist may be overlooked in the ordinary anteroposterior film made primarily to determine the condition of the lungs. In studying the esophagus, in addition to the usual oblique and lateral films, it is desirable to have an anteroposterior roentgenogram of the chest in which, so far as is possible, the detail of structures within the cardiac shadow is brought out.

4. An elongated, tortuous, sclerotic aortic arch, in the absence of aneurysmal dilatation, may produce esophageal compression with symptoms.

5. When a foreign body lodges in the esophagus at the site of a stenosis due to aortic pressure, it should be cautiously dealt with by endoscopic means.

OSTEOGENESIS OF THE HUMAN PERIOTIC CAPSULE *

THEODORE HIERONYMUS BAST, PH.D.
MADISON, WIS.

The petrous portion of the temporal bone and in particular the otic capsule, which stands alone in the category of bones, is of interest from a purely embryologic and anatomic standpoint because of its unique structure, both gross and histologic; but a thorough knowledge of the development and structure of this bone may prove of importance in throwing light on the mystery surrounding otosclerosis and its supposed relation to the dreaded disease, progressive deafness.

The bony otic capsule which houses the sense organs of hearing and equilibrium differs from other bones as to form, structure and function. Bones are conventionally classified as long or cartilage bones and flat or membrane bones. While the otic capsule is classified with cartilage bones, it is neither a long bone nor a flat bone. It is a capsular bone. The cranium is also a bony box, but it is made up of a number of bones which remain as distinct bones, whereas the otic capsule is a bony box made up of a single bone, and as such it is unique.

To the student of bone, the development of flat or membrane bones and long or cartilage bones is well known. In the former, ossification begins at the center and progresses toward the periphery. In the latter, ossification begins in the middle of the shaft as a diaphyseal center and progresses toward the ends, where it ultimately unites with one or more epiphyseal centers at the ends of the long bone. These centers remain more or less distinct throughout life, being separated from each other by epiphyseal lines. In the otic capsule, ossification starts at various points in the cartilaginous capsule, but these centers of ossification fuse so completely that the line of fusion is completely lost.

Histologic examination has revealed to students of bone that the texture of the bone in the otic capsule is different from that of other bones. The most outstanding difference is the normal presence, even in adult otic capsules, of so-called islands of calcified cartilage; not only is there cartilage matrix as is sometimes seen in other bones, but islands of cartilage-like cells are found with their calcified cartilage matrix. The significance of this incomplete replacement of cartilage by bone in this particular bone and not in other bones is still unknown. A study,

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* Presented at the Meetings of the American Otological Society, May 22, 1929.

* From the Department of Anatomy, University of Wisconsin.

therefore, of the early embryonic changes that occur in the formation of the cartilaginous capsule and its transformation into bone seem important for a better understanding of this structure.

Coupled with the purely scientific interest in the histogenesis of the otic capsule is the practical interest which otologists have in the development and structure of this bone in relation to pathologic changes which are associated with various defects of hearing. The problem of otosclerosis in particular has stimulated the members of the American Otological Society to conduct a thorough study of the development, structure and pathology of the otic capsule. It is this society that has made possible the researches on the development of the bony otic capsule on which the article is based.

In this paper I shall limit my observations to the osteogenesis of the bony otic capsule, because Dr. George L. Streeter has already given a thorough account of the development of the cartilaginous capsule in no. 20 of the "Carnegie Contributions to Embryology." It is the aim in this paper to begin where Dr. Streeter's account stops.

HISTOLOGIC OBSERVATIONS

The minute structure of the bone of the capsule has interested many otologists and has resulted in a series of accounts describing it. The most noteworthy of these is the account of Max Meyer (1927), who considered the weblike texture of the framework of the periotic bone as distinctly different from that of other bones. I believe that a knowledge of the process of histogenesis of the capsular bone may help to explain some of these difficulties.

Changes in the Cartilage Preparatory to Ossification.—Siebenmann (1890) said that ossification begins as a result of the entrance of blood vessels into the cartilage which excavate it, and that this, in turn, is followed by deposit of bone. This seems to be the general concept of the beginning of the process of ossification. While blood vessels eventually enter those portions of the cartilage where ossification centers arise, this invasion is by no means the beginning of the process.

According to my preparations, the earliest visible change in the cartilage preparatory to bone formation involves two processes:

1. A marked enlargement of the cartilage cells. The cytoplasm of these cells is at first swollen, and then they shrink away from the matrix. The nuclei become irregular and pyknotic.

2. Concomitant with this cellular change there is a change in the cartilage matrix. It takes the hemotoxylin heavily, appears slightly granular and has a gritty consistency when cut with a knife. There is therefore evidence of an osseous deposit in it (figs. 1 and 2).

Entrance of Osteogenic Buds.—1. The next step is the rupturing of the perichondrium; the ruptured area becomes filled with young connective cells (fig. 3). These cells which resemble phagocytic monocytes seemingly eat their way through the perichondrium and into the enlarged cartilage lacunae, which they fill after removing the necrotic cartilage cells which they contained (figs. 4, 5, 6, 7 and 8). In this manner the changed cartilage described again becomes changed by the entrance of the connective cells into the lacunae. In the changed cartilage on the first step, one sees usually one, sometimes two, necrotic cartilage cells in an enlarged lacuna, but after the entrance of the connective cells each lacuna contains from two to eight cells (fig. 8). The numerous cells contained in each lacuna are not cartilage cells but connective cells which have "wandered in." This fact has been overlooked by most previous workers.

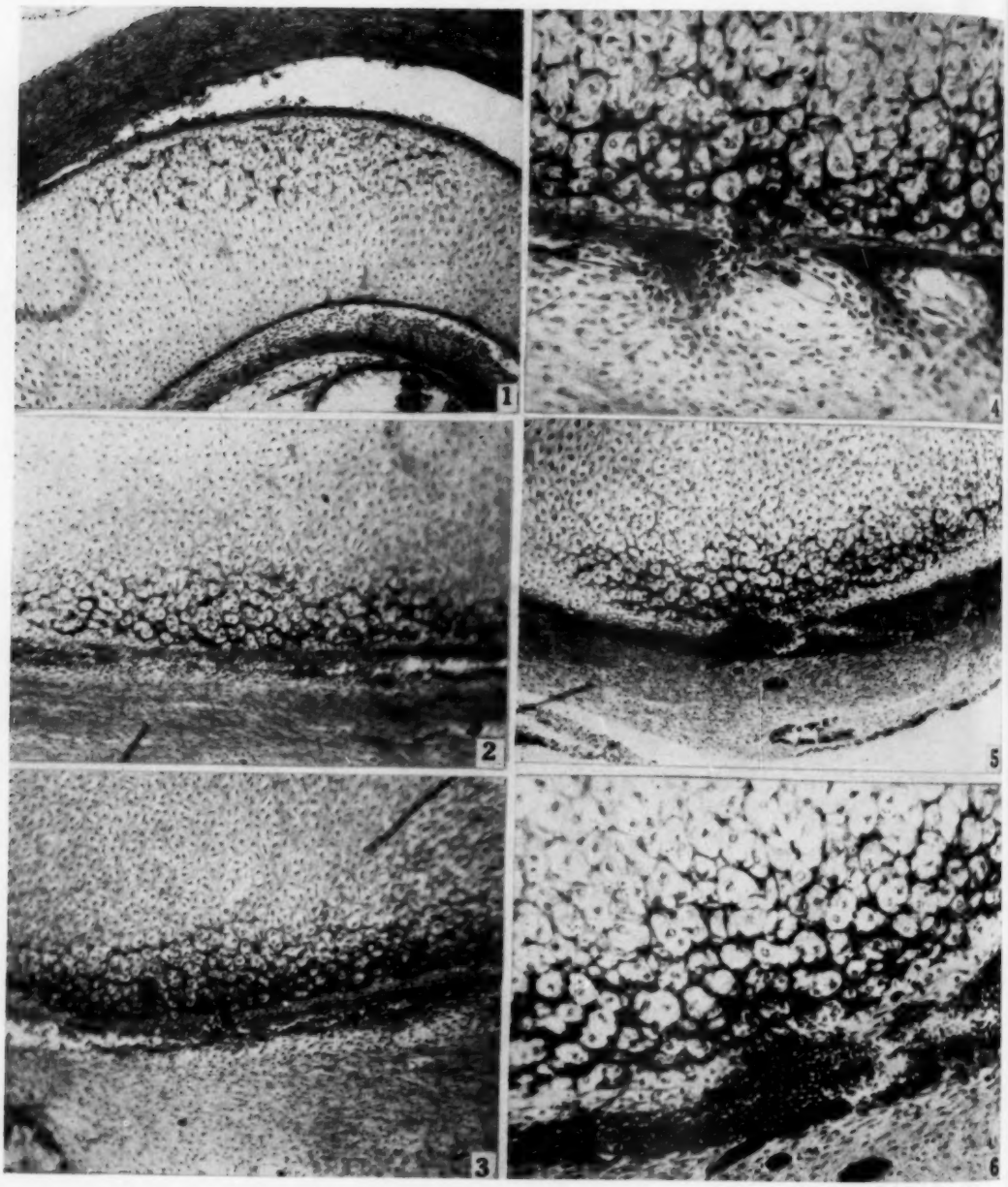
2. Blood vessels now penetrate, through the ruptures of the perichondrium, into this changed cartilage and excavate large channels (fig. 9). Between these channels persist islands of the changed cartilage, which become, through further change, the so-called cartilage islands or interglobular spaces (figs. 10, 11, 12, 13 and 14). In other bones of the body, this excavation is more complete, so that no remnants of cartilage remain except an occasional bit of matrix.

Process of Ossification.—At this stage, three distinct types of ossification begin at approximately the same time.

1. Perichondrial or periosteal bone: This bone is formed from the cambium layer of the perichondrium (this perichondrium becomes periosteum when the cartilaginous periotic capsule is changed to bone) which lines the changed cartilaginous periotic capsule (figs. 9 and 10). The bone thus formed is the same and develops the same as the periosteal bone found in long bones.

Endochondral bone: This bone develops within the excavated channels referred to. It is formed in membrane the same as perichondrial bone, but around the islands of cartilage and in the excavated channels referred to (figs. 11, 12, 13, 14 and 15). This bone, which fills the excavated spaces, is a primary bone and normally remains as such throughout life. In other bones of the body, this primary endochondral bone is usually removed and replaced by secondary or haversian bone if it is replaced at all. It is the structure of this primary bone with its weblike connective tissue framework that attracted the attention of Max Meyer and resulted in his extended treatise on the structure of it. As a matter of fact, it is no different from other primary endochondral bones before its removal and replacement by haversian bone, which is built on a lamellar framework of connective tissue.

PLATE 1



Explanation of Plate 1

Fig. 1 (fetus 38, 147 mm.).—Section through capsule at posterior bend of superior semicircular canal, showing enlarged cartilage cells and a change in the cartilage matrix. This matrix stains deeply with hematoxylin and appears here in black. This change in the cartilage is the earliest step in the development of an ossification center. Reduction approximately one-third off from a magnification of × 72.

Fig. 2 (fetus 38, 147 mm.).—Section through the otic capsule below and slightly anterior to the internal auditory meatus. Part of the cartilaginous capsule, together with its perichondrium, is pictured showing an early ossification center. The cartilage next to the perichondrium shows a change similar to that in figure 1, but at a slightly more advanced stage. Reduction approximately one-third off from a magnification of × 72.

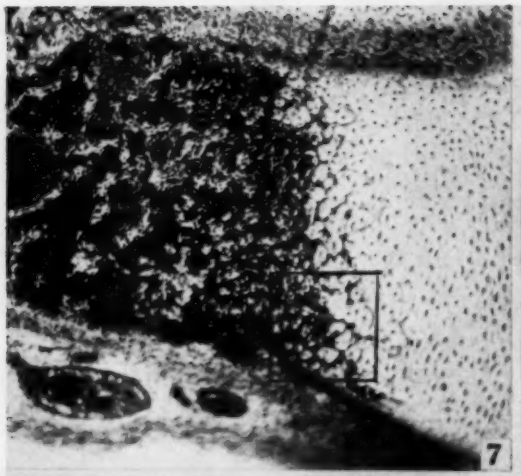
Fig. 3 (fetus 38, 147 mm.).—Section through otic capsule between the base of the cochlea and the dura. The inner part of the capsule is shown. The change in the cartilage under the periosteum is more advanced than in figure 2. In this picture the periosteum is ruptured by the entrance of an osteogenic bud. The gap in this periosteum is filled with invading cells. Reduction approximately one-third off from a magnification of × 72.

Fig. 4 (fetus 38, 147 mm.).—Section through the upper medial wall of the internal auditory meatus. This center of ossification is more advanced than that in figure 3. The gap in the perichondrium is larger, and the invading cells have penetrated it and are invading a few of the cartilage lacunae. A small blood vessel is entering the gap in the perichondrium but has not entered the cartilage. The two dark areas on each side of the gap in the perichondrium are the first anlagen of perichondrial bone. Reduction approximately one-third off from a magnification of × 158.

Fig. 5 (fetus 38, 147 mm.).—Section through the lateral wall of the internal auditory meatus. The gap in the perichondrium is larger than that in figure 4 and is packed with invading cells. These cells have filled a group of cartilage lacunae. Reduction approximately one-third off from a magnification of × 72.

Fig. 6.—The same area shown as figure 5 but at a magnification of × 158. This shows more clearly the invading cells within the lacunae of cartilage.

PLATE 2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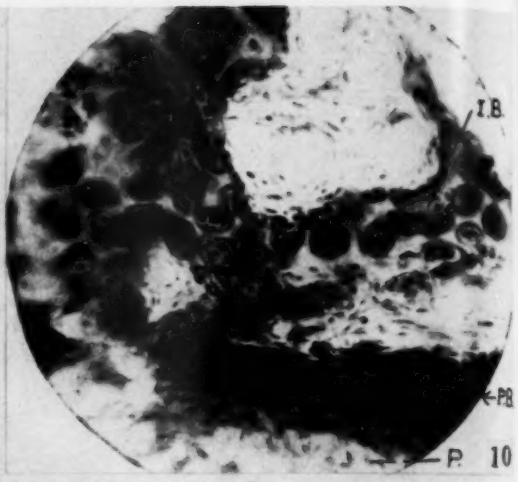

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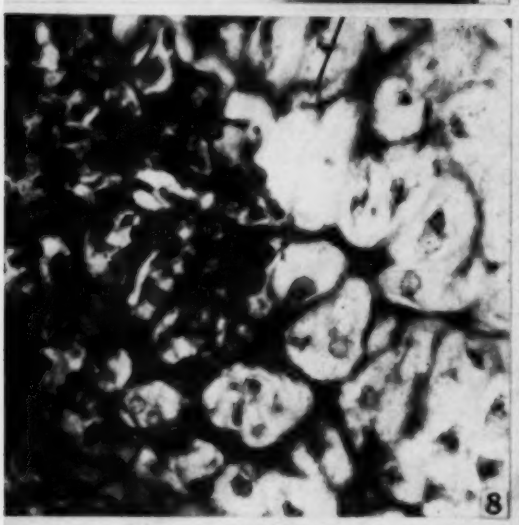
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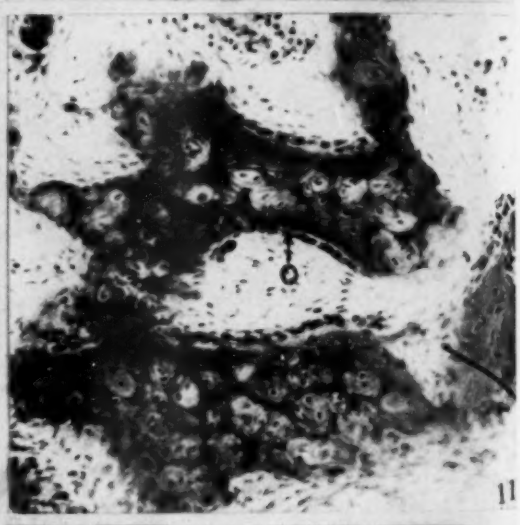

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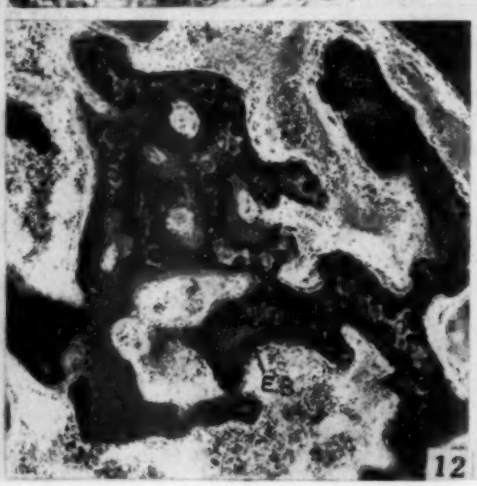

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Explanation of Plate 2

Fig. 7 (fetus 38, 147 mm.).—Section through the otic capsule at the facial canal, showing the edge of a center of ossification advancing into the remaining cartilage. The area indicated by the square is enlarged in the next figure. Reduction approximately one-third off from a magnification of × 72.

Fig. 8.—Enlarged view of the square marked in figure 7. At the right are seen the enlarged lacunae of cartilage containing one cell each and the changed cartilage matrix between them, while at the left the cartilage cells are destroyed and the lacunae filled with the invading connective cells. These invading cells within the lacunae became osteoblasts, filling the lacunae with bone. In the following illustrations, the nests of cells in the ossified otic capsule are the cells which invaded the lacunae and were then changed into bone cells. This bone formed within the lacunae is spoken of as intrachondral bone. Reduction approximately one-third off from a magnification of × 264.

Fig. 9 (fetus 13, 161 mm.).—Section of a portion of the capsule of the vestibule showing how the cartilage becomes excavated by the osteogenic buds, leaving, however, islands of the changed cartilage described in previous figures. *C* indicates cartilage; *I.B.*, islands of changed cartilage which are being changed into intrachondral bone by the calcification of the cartilage matrix and the metamorphosis of the "wandered-in" connective tissue cells within the lacunae into bone cells; *P.B.*, perichondrial bone, and *P*, periosteum. Reduction approximately one-third off from a magnification of × 24.

Fig. 10.—The same area as that indicated by circle in figure 9. *I.B.* indicates developing intrachondral bone; *P.B.*, perichondral bone, and *P.*, periosteum. Reduction approximately one-third off from a magnification of × 136.

Fig. 11 (fetus 21, 183 mm.).—Developing intrachondral bone which is more advanced than that in figure 10. On the surface of this bone and lining the excavated areas at *O.*, a row of osteoblasts is beginning to lay down endochondral bone. Reduction approximately one-third off from a magnification of × 130.

Fig. 12 (fetus 2, 230 mm.).—Portion of the otic capsule showing intrachondral bone with a small amount of endochondral bone at *E.B.* Reduction approximately one-third off from a magnification of × 60.

Intrachondral bone or the so-called calcified cartilage or cartilage islands: The islands of changed cartilage mentioned which remained, owing to incomplete excavation by the vascular osteogenetic buds, have always attracted the attention of otologists and students of bone. Most authors (Böttcher, Moos and Steinbrügge, Manasse and Shin-izi-Ziba) have considered these as the product of cartilage metaplasia into bone. The only dissenting opinion was that of O. Mayer, who stated that all cartilage cells were replaced by bone cells. Although in my earlier papers (1927 and 1928) I expressed the view that the islands were the results of cartilage metaplasia, my completed observations agree with those of Mayer. While these cartilage islands retain the general appearance of calcified cartilage, a careful study of the process shows that the cells of the osteogenic buds penetrate the cartilage matrix and thus invade the lacunae. Blood vessels do not follow into these islands. These invading cells then become bone cells and deposit bone around themselves within the lacunae. Thus, in each cartilage lacuna of the islands one can see from two to eight or even more bone cells as compared with the single cartilage cell found in a typical cartilage lacuna. This bone I have called intrachondral bone (figs. 10, 11 and 12). I realize that the meaning of this term is almost the same as endochondral. But I believe it is generally understood that the term endochondral is a misnomer for the bone to which it is applied, for the so-called endochondral bone is not formed in cartilage but rather in membrane in a place which was previously occupied by cartilage.

Do Bone Cells Remain Active?—In 1921 and 1923, I presented evidence that bone cells remained active within bone, and at least in young bone retained the power of division by the amitotic method, thus causing expansion of bone from within. In the capsule of the internal ear, this phenomenon was again observed. One can see numerous bone cells the nuclei of which are in the process of amitotic division (fig. 19). The cell cytoplasm probably never completely divides, for one finds that all bone cells are connected to other bone cells by means of protoplasmic processes. It is because of the network of the protoplasmic processes that expansion and bone deposit can go on between and around the individual bone cells.

The Fissula Ante Fenestram or Cozollino's Zone.—The fissula ante fenestram or Cozzolino's zone has been described for the various fetal and postnatal ages by Perozzi in 1920-1921, and by Kosokabe in 1922. Their work was based on sections only. My description is based on serial sections and on wax and plaster of paris reconstructions. The preparations show that the shape or form of this structure varies with the individual, but its histologic structure and its continuity with the

vestibule and middle ear are constant. The structure is an evagination from the vestibule at the point where the vestibule and scala vestibule meet. From my models it is apparent that the site of its communication with the vestibule is quite constant, while its communication with the middle ear varies. In some cases this communication is slitlike, extending medially from the anteromedial niche of the oval window. In other cases the communication with the middle ear is an oval area in the region of the tendon of the tensor tympani.

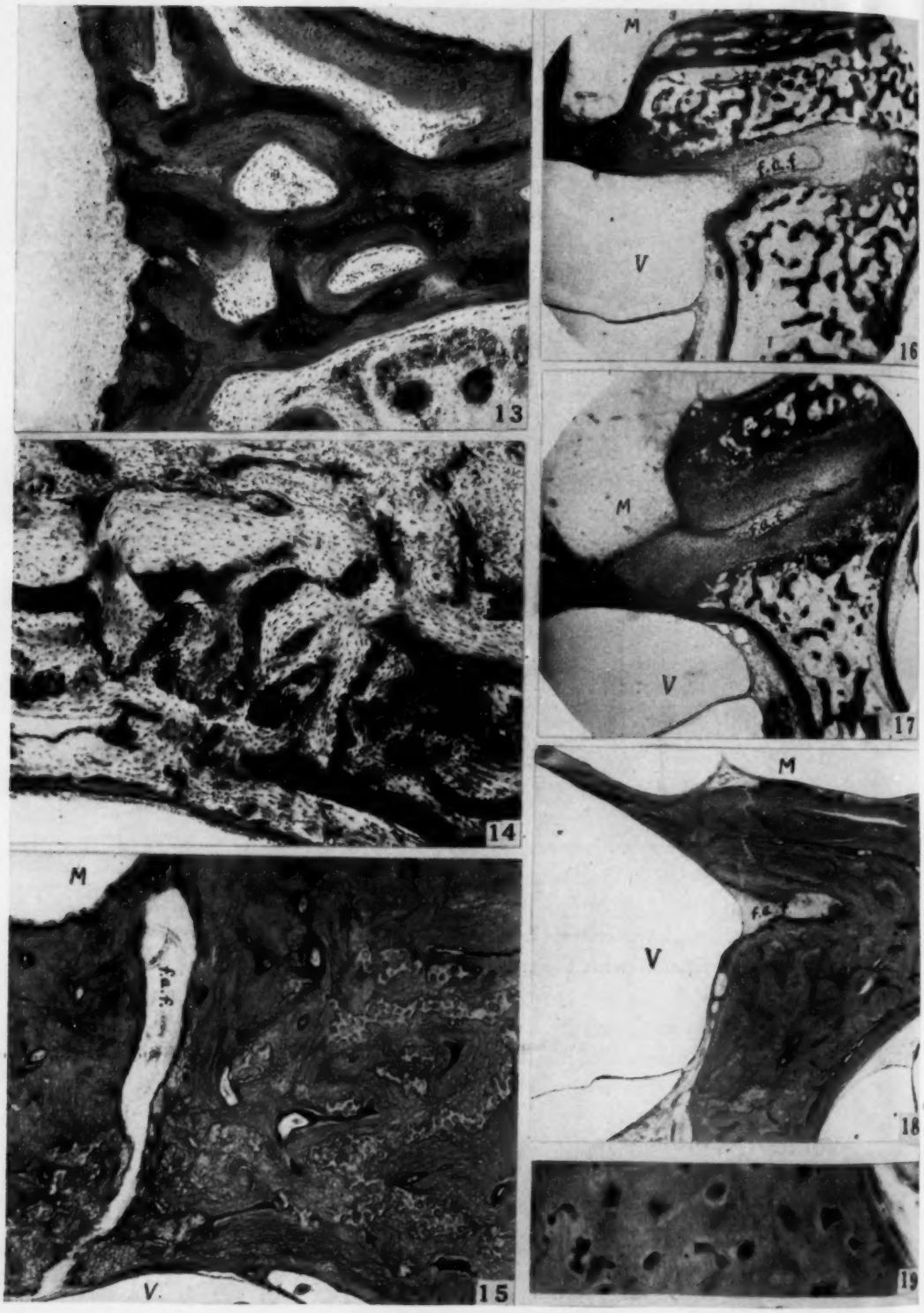
Histologically, the fissula is a fissure-like space filled with a loose connective tissue (figs. 15, 16, 17 and 18). I cannot agree with previous statements that it is filled with a dense fibrous tissue; it consists of a loose connective tissue which is continuous with the very loose periotic or perilymphatic connective tissue. At the side next the middle ear, the fissula receives several small blood vessels which proceed into it for only a short distance and then break up into capillaries. The portion toward the vestibule is free from blood vessels. Toward the side next the middle ear, the connective tissue is of a denser type than that toward the vestibular side. The density of this connective tissue increases with age but hardly to the extent that it could be considered as perichondrium. Neither is there any histologic evidence that growth takes place at this point, as suggested by Perozzi.

Up to the present time, my studies have presented nothing conclusive as to the function or meaning of the fissula ante fenestram. Around this fissula the capsule remains cartilaginous until birth, and in many cases throughout life.

The Fossula Post Fenestram.—In a small percentage of the fetal ears observed, there has been noted an evagination of the vestibule into the periotic bony capsule posterolateral to the niche of the round window. Histologically, the tissue filling this evagination is much the same as that of the fissula ante fenestram. This fossula has never been found to extend through the capsule to the middle ear, although Ibsen describes this communication with the middle ear in a very small percentage of cases.

In my preparations, the tissue of the fossula becomes continuous with the bone-marrow of the bony capsule. This relation of the bone marrow with the loose connective tissue of the fossula, which in turn is continuous with the perilymph or periotic spaces, suggests that the fossula is a possible area of infiltration for the periotic fluid. If the fissula ante fenestram and the fossula post fenestram are both evaginations of the vestibule, this suggestion may apply to both. Experiments should be devised to test this hypothesis.

PLATE 3



EXPLANATION OF PLATE 3

Fig. 13 (fetus 2, 230 mm.).—Portion of the otic capsule showing the islands of intrachondral bone surrounded by a considerable amount of endochondral bone. Reduced approximately one-third off from a magnification of $\times 60$.

Fig. 14 (fetus 16, 363 mm.).—Section of the otic capsule of a full term fetus, showing the intrachondral and endochondral bone. Reduction approximately one-third off from a magnification of $\times 72$.

Fig. 15.—Autopsy from the Johns Hopkins Ear Institute. The left ear of a white man, aged 75 years. Section through the capsule anterior to stapes, showing the persistence of intrachondral bone or the so-called interglobular spaces. The slit at *f.a.f.* is the fissula ante fenestram which extends from the vestibule *V.* to the middle ear *M.*

Fig. 16 (fetus 21, 183 mm.).—Horizontal section through a portion of the internal ear anterior to the stapes showing the fissula ante fenestra (*f.a.f.*) at the level where it opens into the vestibule (*V.*). *M.* indicates the middle ear. Reduction approximately one-third off from a magnification of $\times 31.5$.

Fig. 17.—The same fetus as that shown in figure 16, but at a higher level where the fissula ante fenestram (*f.a.f.*) communicates with the middle ear (*M.*). *V.* indicates the vestibule.

Fig. 18.—Autopsy from the Johns Hopkins Ear Institute. Section through the ear of a white man, aged 40 years. The region is similar to that shown in figure 16. This shows the persistence of the fissula ante fenestram (*f.a.f.*). *V,* indicates the vestibule, *M.* the middle ear.

Fig. 19 (fetus 21, 183 mm.).—Section through primary endochondral bone of the periotic capsule, showing amitotic division of bone cells. Reduction approximately one-third off from a magnification of $\times 650$.

SUMMARY

1. The bone of the periotic capsule is of three types: (*a*) perichondral bone, (*b*) endochondral bone and (*c*) intrachondral bone or bone formed within the lacunae of the calcified cartilage matrix which persists.

2. The process of ossification of the cartilaginous capsule begins as follows: (*a*) enlargement of the lacunae of cartilage; (*b*) a modified staining reaction of the cartilage matrix; (*c*) the entrance of connective tissue cells into the enlarged lacunae which replace the cartilage cells; (*d*) the entrance of vascular osteogenic buds which excavate large channels in this changed cartilage; this excavation is not complete, thus leaving islands of cartilage; (*e*) primary endochondral bone is formed in the excavated parts; (*f*) the connective tissue cells which wandered into the lacunae of the islands of cartilage change into bone cells and surround themselves with bone; the cartilage matrix of these islands is calcified, thus producing a peculiar type of bone which has been designated as intrachondral bone, and (*g*) perichondral bone is formed around the original cartilaginous capsule.

3. At the junction of the vestibule with the scala vestibuli there is a pouching-out of the vestibule, which is filled with perilymphatic tissue. This tissue extends from the vestibule to the middle ear. With age this tissue becomes denser. Around it the cartilaginous capsule persists even into adult life. This area is variously konwn as fissula ante fenestram (Siebenmann) or Cozzolino's zone (Perozzi).

4. In a small percentage of the fetuses observed, a similar pouching-out of the vestibule was seen just posterolateral to the oval window. This evagination ended blindly in the bony capsule, where it became continuous with the bone-marrow. This structure is known as canalis membranaceus caecus (Ibsen) or as fossula post fenestram (Siebenmann).

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THE EFFECT OF AUDIBLE AND OF SUBAUDIBLE VIBRATIONS ON THE ACUITY OF HEARING*

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This paper presents the results of studies which were continued in connection with the subject of paracusis,[1] and especially an extension of those studies into the field of subaudible vibrations. Briefly, our tests have indicated the following: 1. No one hears better in the presence of a noise. 2. No one hears better in the presence of subaudible vibrations. In other words, both audible and subaudible vibrations, under all conditions, fail to increase the acuity of hearing.

It would seem that this subject has a definite bearing on clinical practice, in that for the past few years many methods based on the use of audible and inaudible vibrations have been advanced and used clinically for the improvement of hearing. As somewhat favorable statements have been made as to the value of such methods of treatment, it seemed to us that it would be helpful to put the matter to the test of precise measurement. Many clinicians have been of the opinion that vibration can improve the hearing, by exercising the end-organ. In different countries recommendations have been made for the use of audible, subaudible and superaudible vibrations for providing this exercise. In Russia, recent work has utilized the audible range; apparatus for this purpose was recently exhibited at the All-Russian Congress of Zoology, Anatomy and Histology, where it attracted considerable interest. From Germany come reports of the utilization of the superaudible frequencies—30,000 vibrations per second—for stimulating the cochlea.[2] In this country, the most interest has centered in the utilization of subaudible vibrations. Instruments have been employed for creating gross vibrations analogous to the sound waves, but of a frequency below the threshold of hearing; that is, below 20 vibrations per second.

In our earlier work on this subject, it was found that audible vibrations of any frequency or intensity reduced the acuity of hearing in

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1. Knudsen, V. O., and Jones, Isaac H.: Paracusis, Laryngoscope **35**:817 (Sept.) 1926. Shambaugh, G. E.: Explanation for Symptoms of Paracusis Willisi; A Demonstration, Arch. Otolaryng. **6**:228 (Sept.) 1927.

2. Science **69**:12 (May 31) 1929.

all persons, regardless of the presence or absence of any type of defect in hearing. Those with an impairment of the conductive mechanism who have considered that they heard better in the presence of a noise were found to constitute no exception to this general observation. In other words, it appeared that there has been an inaccuracy in the conception that certain people who are hard-of-hearing hear better in the presence of a noise. The person with normal hearing, the person with impaired perception, and the person with impaired conduction, all heard less well in a noise. It was found that the one with impaired conduction, when subjected to a standard noise, suffered less loss of acuity than the one with impaired perception or the one with normal hearing. The person with impaired perception and the person with normal hearing were markedly affected by the noise, whereas the person with impaired conduction showed only a relatively slight decrease in the acuity of hearing. When these three types of patients were subjected to a noise which to them was equally loud, they all sustained approximately the same loss of acuity; whereas, when they listened to the same physical noise, the one with normal hearing and the one with defective perception suffered much more than the one with defective conduction.

This is readily accounted for in that the noise is more or less shut off from the cochlea of the person whose conductive mechanism is poor by the fact of that defect. In contrast, the normal person suffers the full disturbing effect of the noise because his normal conductive mechanism transmits the full intensity of it to the cochlea. The person whose perception is impaired experiences a disturbing effect because of his normal conductive mechanism; in addition, the noise is more disturbing to the impaired cochlea than it is to a normal cochlea. Again, because of the effect of the noise on the cochlea, the normal person attempts to speak above the noise, and, therefore actually elevates the loudness of his voice. The person with lowered perceptive function also elevates the loudness of his voice, not only in the presence of a noise, but at all times, for the simple reason that he does not hear his own voice unless he speaks loudly. In the same physical noise, therefore, the normal person and the person with impaired perception speak relatively louder than the person with impaired conduction. For the reasons just enumerated, the one with poor conductive power experiences a relative advantage when conversing in a noise with a normal person or one whose perception is impaired. The noise provides a favorable environment for the poorly conductive ear, so that the person is more nearly on a par with the normal hearer, and consequently it is natural that he should believe that his own hearing has been improved by the noise. Actually, however, provided he hears the noise, he has less acuity of hearing in the noise than he has in the quiet. It is not necessary,

therefore, to consider that such a person presents an anomaly in the physiology of hearing.[3] He conforms to the general rule; namely, that all persons hear less acutely in the presence of a noise.

The loss of acuity of hearing in the presence of a noise can be accounted for on the basis of auditory masking, which is caused by the interfering effect of tones or noise upon the hearing of other tones or speech. Wegel and Lane [4] showed how the presence of one tone interferes with, or masks, the hearing of another. It was found that any tone interferes with the hearing of any other tone, and it was especially noted that the presence of low-pitched tones interferes greatly with the hearing of higher-pitched ones; also that there is marked masking of tones in close proximity to the masking tone. One of us (V. O. K.) investigated the masking effect of noise on the hearing of speech.[5] This work shows that a noise, however slight, interferes with the hearing of speech, and that the interfering effect increases as the loudness of the noise increases. This applies to persons with normal hearing and to those with impaired conduction or perception.

Therefore, as audible vibrations produce this masking effect, the question arises whether there is not some beneficial effect owing to the vibration, that is greatly offset by the masking effect. In order to determine whether the gross vibrations of the drum membrane and ossicles could, per se, increase the hearing acuity of the person with defective conduction, experiments were undertaken in which auditory masking could not possibly play a part; that is, this type of patient was subjected to gross, but subaudible, vibrations. It seemed that in this way it would be possible to find out, once and for all, whether the acuity of hearing can be improved by any type of external vibration. It is noteworthy that the claims of those who hear better in a noise are nearly always associated with the noise in a moving vehicle, as an automobile, a subway train, a trolley car or other noisy conveyance. It is known that the vibrations produced by these moving vehicles are especially rich in low frequency vibrations, even subaudible vibrations. This circumstance emphasizes the advisability of determining whether the subaudible vibrations actually do have a beneficial effect on hearing.

DESCRIPTION OF APPARATUS

In an attempt to devise satisfactory apparatus for producing subaudible vibrations, various devices were designed and some of them were constructed and

3. Turner, Logan: Diseases of the Nose, Throat and Ear, pp. 268 and 349.

4. Wegel, R. L., and Lane, C. E.: Auditory Masking of One Pure Tone by Another and Its Probable Relation to Dynamics of the Ear, Physical Rev. **23**:266 (Feb.) 1924.

5. Knudsen, V. O.: Interfering Effect of Tones and Noise Upon Speech Reception, Physical Rev. **26**:133 (July) 1925.

tried out; among them a large vacuum-tube oscillator operating a powerful loud-speaker. Tests were made with this apparatus, but it was felt that a more powerful source of vibrations should be provided. Such a vacuum-tube apparatus is satisfactory in principle, but requires prohibitively bulky and expensive equipment in order to produce vibrations of sufficient intensity for the purpose of these tests. Several other devices were considered, including a piston or plunger connected with an airtight chamber; apparatus for producing pneumatic vibrations in the canal of the ear; a cone type loud-speaker driven by an electric motor and worm gear attached to an eccentric. These devices were abandoned in favor of a simpler

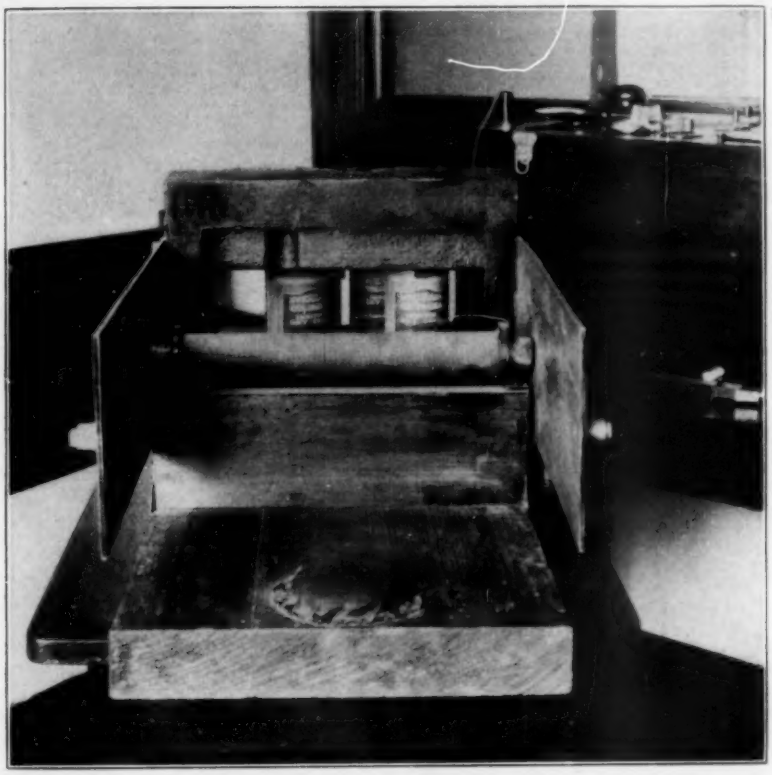

Fig. 1.—Vibrator used for producing subaudible vibrations.

and more practical device that produces an amplitude of vibration ten times greater than that produced by any of the other devices. This is the apparatus which was used in obtaining the data contained in this report. A photograph of the apparatus is shown in figure 1. It consists essentially of two steel vanes, each vane being 10 cm. wide, 33 cm. long and 0.3 cm. thick. The two vanes are mounted vertically to a massive frame, and are separated horizontally by a distance of 26 cm., sufficiently wide for the head to be placed between the vanes, and each vane to be then in close proximity to each ear. A chin rest mounted on the frame maintains the head in the proper position. The steel vanes are set in oscillation by an electromagnet, which causes the vanes to vibrate in their natural

frequency, like a tuning fork actuated electrically. The frame is sufficiently heavy to eliminate any adventitious vibration, and it keeps the entire apparatus in a fixed position. An electromagnet, containing a soft iron core, is placed between the two vanes, so that the iron core almost touches both vanes (fig. 1). The circuit which actuates the electromagnet is interrupted by the vibration of one of the vanes which makes intermittent contact through tungsten points. The noise of the make and break at the contact point is eliminated by immersion of the contact points in oil. The vanes are adjusted to such a length as to give a natural rate of vibration of 16 per second. The amplitude of vibration of the vanes is controlled by means of a variable resistance in the electric circuit, and by means of the closeness of adjustment of the contact points. These two means of adjustment proved satisfactory for obtaining any desired amplitude of vibration. The amplitude of vibration of the vanes can be varied continuously from zero to 1.7 cm., measured at the distal ends of the vanes. For vibrations having an amplitude in excess of 1 cm., the pneumatic vibrations in the canal of the ear are sufficient to produce an appreciable sensation of pressure in the middle ear. When the head is in position between the vanes, the variations of pressure in the air surrounding the ears, and therefore in the auditory canals, amounts to at least one part in a hundred. This is ten million times the variation of pressure required to produce a barely audible tone at a frequency of 1,000 double vibrations. Such an intense variation of the pressure in the auditory canal produces an appreciable movement of the drum membrane and ossicular chain. The amplitude of vibration of the drum membrane produced by this apparatus is about 0.1 mm. This is sufficient to produce the sensation of movement without being sufficiently gross to produce the sensation of pain. This amplitude is about one hundred times the amplitude required to elicit the sensation of vibration.[6] It would seem, therefore, that the vibration communicated to the drum membrane by this apparatus would be ample to simulate the subaudible vibrations that accompany ordinary noises or even loud noises. The vibrations produced in the two ears are the same; that is, both drum membranes are caused to move inward at the same time and outward at the same time. In other words, the vibrations reaching the two ears are in phase.

METHOD OF TESTING

The methods of conducting the tests were described in detail in the previous report on paracusis.[1] The hearing acuity of the person under test was measured with the audio-amplifier[7] before, during and after exposure to the subaudible vibrations. Tests were made with the subaudible vibrations at two different intensities, corresponding to amplitudes of vibration of the vanes, of 2 and 10 mm., respectively. The time of exposure was varied from five to thirty minutes, in different tests. In order to test the acuity of hearing in the presence of these subaudible vibrations, especially constructed frames held the telephone receivers about 1 cm. from each auricle. There were large perforations in the frame surrounding the receiver, so that the pressure waves in the air could communicate freely with the external canals and drum membranes. In this way, the person under test could listen to the tones produced by the audio-amplifier, and yet be exposed to the gross subaudible vibrations.

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6. Knudsen, V. O.: Hearing with Sense of Touch, J. Gen. Psychol. **1**:320 (April) 1928.

7. Knudsen, V. O., and Jones, I. H.: Functional Tests of Hearing, Laryngoscope **34**:673 (Sept.) 1924.

Tests of speech articulation, similar to those described in the paper on paracusis, also were conducted before, during and after exposure to the subaudible vibrations. By these two types of tests, the patient's acuity of hearing was tested, both for tones and for the conversational voice.

RESULTS OF TESTS

Three persons were selected for the tests, for the following reasons; each patient had a marked bilateral lesion of the conductive mechanism; each patient had always considered that the acuity of hearing was much better in a noise, particularly in a noisy moving vehicle; all three were intelligent, interested and eager to cooperate. Two of the three patients were physicians, one an otologist. The third, a woman, was particularly interested in the tests because of her faith in the value of subaudible vibrations for the "hard-of-hearing."

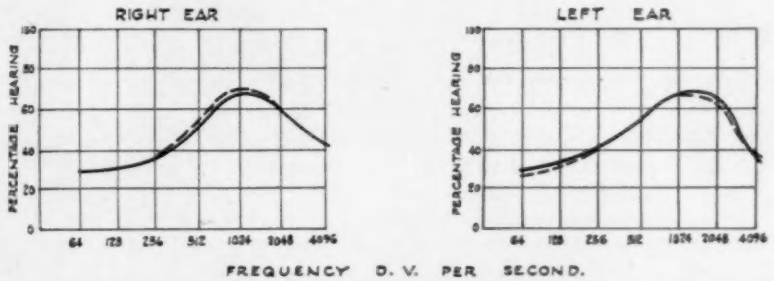

Fig. 2.—Curves showing the acuity of hearing of a person with paracusis (Dr. R.). In this and the following figures the solid line gives the percentage of hearing for tones in the quiet. The dotted line gives the acuity of hearing in the presence of subaudible vibrations. The amplitude of vibration of the vanes was 1.1 cm.

Each patient was subjected to subaudible vibrations of two or more different intensities, for the purpose of determining whether any particular degree of vibration might be more helpful than another.

A typical set of audiograms is exhibited in figures 2, 3 and 4. In each chart, the solid line shows the percentage of hearing for tones when the subject was listening in the quiet. The dotted line shows the percentage of hearing when the subject was listening in the presence of a subaudible vibration corresponding to an amplitude of vibration of the vanes of 1 cm. This intensity of vibration was sufficient to be felt by the person under test; that is, the patient experienced a sensation of pressure against the drum membrane. As the charts indicate, there is no significant difference between the acuity of hearing in the quiet and that in the presence of the subaudible vibrations. In fact, if there is a difference, the data indicate a slightly diminished

acuity in the presence of subaudible vibrations, although the difference is no greater than could be attributed to error.

In all cases, the acuity of hearing was also measured in the presence of a less intense vibration—that corresponding to an amplitude of the vanes of 0.1 cm. The measurements of the acuity of hearing in the presence of this diminished vibration did not differ from the measurements of the acuity in the quiet or in the presence of the more intense vibrations. If the data were plotted in figures 2, 3 and 4, the curves would practically coincide with those already shown. In two cases, the

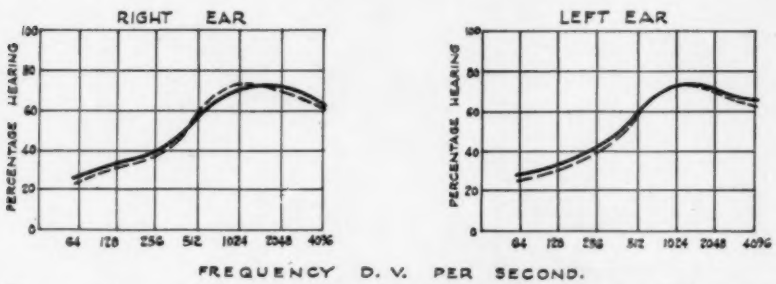

Fig. 3.—Curves showing the acuity of hearing of a person with paracusis (Dr. T). The amplitude of vibration of the vanes was 1.1 cm.

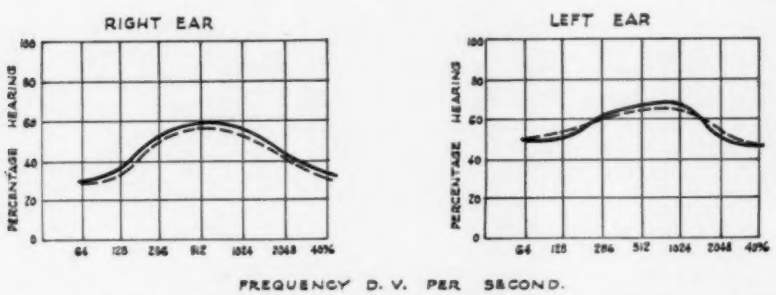

Fig. 4.—Curves showing the acuity of hearing of a person with paracusis (Mrs. C.). The amplitude of vibration of the vanes was 1 cm.

vibration was varied from a barely perceptible amplitude of vibration to an amplitude of 1.7 cm. There was no degree of vibration that provided any measurable improvement in the acuity of hearing. All these data seem to warrant the conclusion that no degree of subaudible vibration, having a rate of 16 double vibrations a second, provides an improvement in the acuity of the hearing of tones.

Additional tests were conducted using an electromagnetic cone speaker driven by an eccentric at a rate that could be varied from 12 to 20 vibrations a second. Measurements obtained in the presence of these subaudible vibrations were in agreement with those obtained at a frequency of 16 double vibrations. It would seem, therefore, that no

degree of subaudible vibrations between frequencies of 12 and 20 per second has any measurable effect on the acuity of hearing.

Tests also were conducted on two of these persons fifteen minutes after they had been subjected to subaudible vibrations for thirty minutes. Again there was no evidence of a change in the acuity of hearing owing to the treatment with subaudible vibrations.

The tests of the hearing of speech in the presence of subaudible vibrations, like the tests of the acuity of hearing of tones, gave negative results. That is, there was no evidence that the hearing of speech was facilitated by any degree of subaudible vibrations. Tests of speech articulation were conducted in the quiet and repeated when the person

Results of Tests of Speech Articulation

Person Tested	Articulation in Quiet, per Cent			Articulation in Presence of Subaudible Vibrations, per Cent		
	Vowels	Consonants	Syllables	Vowels	Consonants	Syllables
Dr. R.	91	42	19	89	37	17
Mrs. C.	74	29	8	78	28	8
Dr. T.	92	47	22	94	46	21

was subjected to subaudible vibrations of the same magnitude as those used for the measurements which are represented in figures 2, 3 and 4; that is, the amplitude of vibration of the vanes was 1 cm. In all tests, the patient's head was in the same position between the vanes of the vibrator.

A summary of the results of the tests of speech articulation is given in the accompanying table. The results indicate that for the hearing of speech no benefit was derived from the subaudible vibrations. In fact, there is some indication that the subaudible vibrations actually interfered with the hearing of speech, although the differences are no greater than those which may be attributed to error.

It appears, therefore, that neither the acuity of the hearing for tones nor the acuity of the hearing for speech receives any benefit from subaudible vibrations.

STENOSIS OF THE NASOPHARYNX*

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Cicatricial stenosis of the nasopharynx occurs rarely. In the past the condition has been difficult to treat, as evidenced by the great variety of procedures advocated for its relief. Many of the proposed operations are of little practical value. A few, when properly carried out, will restore satisfactorily the nasopharyngeal lumen. Most of the reports in the literature give little clinical data, especially as regards end-results. Eighteen cases of nasopharyngeal stenosis have been observed in the Mayo Clinic.

Although stenosis of the nasopharynx is usually acquired, a few cases of true congenital nasopharyngeal stenosis, not including atresia of the choanae, are reported in the literature. Two cases of this type, reported by Hall,[1] are of particular interest because of the ease with which the abnormalities were corrected. The patients were children, aged 8 and 10 years, respectively. Atresia between the soft palate and the posterior wall of the nasopharynx was readily freed with the finger and did not show any tendency to recur. Wassermann,[2] Bailey[3] and Mackenty[4] each reported a case of this type, and one of the cases reported here should be added to this group. In the cases of acquired stenosis of the nasopharynx, syphilis is generally conceded, by the various writers on the subject, to be the most common cause. In fact, in a group of sixty-nine cases reviewed by Wright and Smith[5] in 1914, fifty-eight were said to be due to syphilis. These figures vary so strikingly from observations in the clinic that I cannot but wonder whether a large proportion of these cases were not seen in charity clinics, where the patients, because of their own indifference or

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* From the Section on Laryngology, Oral and Plastic Surgery, the Mayo Clinic.

* Read before the American Laryngological, Rhinological and Otological Society, San Francisco, July 4 to 6, 1929.

1. Hall, F. J. V.: Adhesion of Soft Palate to Naso-Pharynx, Brit. M. J. **1**:18, 1909.

2. Wassermann, quoted by Bailey: J. Iowa M. Soc. **7**:57, 1917.

3. Bailey, F. W.: Occlusion of the Naso-Pharynx, J. Iowa M. Soc. **7**:57, 1917.

4. Mackenty, J. E.: Nasopharyngeal Atresia, Tr. Am. Laryngol. Ass. **48**: 131, 1926.

5. Wright, Jonathan; and Smith, Harmon: A Text-Book of the Diseases of the Nose and Throat, New York, Lea & Febiger, 1914, pp. 683.

ignorance, failed to secure treatment during the stage of active pharyngeal ulceration. The next most common etiologic factors mentioned by Wright and Smith, in order of their frequency, are trauma, diphtheria, tuberculosis, congenital anomalies and inflammatory lesions.

OBSERVATIONS AT THE MAYO CLINIC

In the patients seen in the clinic, the most common cause of the condition was trauma incident to the removal of tonsils and adenoids. This appeared to be the direct cause of the stenosis in six cases. Four of the six patients were children aged less than 12 years, who had had their tonsils and adenoids removed. The two remaining patients in the group had had simple tonsillectomy. One of these patients was 37; the other was 52. The interval between the time of the operation and the onset of symptoms varied from a few weeks to two years. The latter period may well lead to skepticism concerning the probable relationship between the tonsillectomy and the stenosis; yet no other likely cause could be determined. Syphilis was considered responsible for the condition in five cases, although in one of these the patient said that the stenosis had developed following diphtheria at the age of 9 years. In this, as well as in two other cases, the syphilis was hereditary, and the nasopharyngeal stenosis was of long duration, varying from ten to seventeen years. On the other hand, in the two cases of acquired syphilis, the condition in the throat had been present for only one year. In only one case, aside from the aforementioned one with hereditary syphilis, was diphtheria given as the direct cause of the scarring and contraction. Unfortunately, the Wassermann reaction was not recorded in this case. However, the patient, a child, aged 7, did not show definite evidence of inherited syphilis. In one patient the stenosis occurred at the age of 11 following an acute inflammatory condition of the throat, the etiology of which was not known. In another, the condition was due to an indeterminate granuloma which was suggestive, clinically, of rhinoscleroma, although the Frisch bacillus was never demonstrated. Investigation for syphilis was negative in both of these cases. The stenosis apparently was congenital in one case. In another case it had resulted from drinking sulphuric acid by mistake, three weeks prior to examination in the clinic. Marked scarring and contraction of the oropharynx, hypopharynx and esophagus due to the acid were present. The esophageal obstruction was marked, and the dilation of this only has been carried out to date. In none of the articles on stenosis of the nasopharynx, which have come to my attention, is mention made of the possibility of rhinoscleroma being an etiologic factor. Two cases in the group seen in the clinic were due to this disease. The infection, in fact, produces not infrequently characteristic concentric stenosis of the nasopharynx, often of marked degree. The nasal obstruction so often

present in these cases, however, is usually due to the granulomatous mass, or to stenosis present in the nasal fossae. In addition, the soft palate is often retracted and elevated, and the uvula may be completely destroyed (figs. 1 to 7).

The etiology in the cases seen in the clinic and the number of cases in each etiologic group are as follows: tonsillectomy and adenectomy, four; tonsillectomy alone, two; hereditary syphilis, three; acquired syphilis, two; indeterminate inflammatory process, two; rhinoscleroma, two; diphtheria, one; caustic (sulphuric acid), one, and congenital abnormality, one.

Fig. 1.—Cicatricial stenosis of the nasopharynx following tonsillectomy; before treatment.

The age at which these patients came for examination is not of particular significance since with few exceptions they all had been under treatment previously for the condition. Most of them had been operated on from one to several times, in an attempt to open the contracted nasopharynx. A consideration of the age at which the stenosis developed, however, is of considerable interest. Stenosis developed in the first decade in seven cases (38 per cent), in the second in three cases (16 per cent), in the third in three cases (16 per cent), in the fourth in two cases (11 per cent) in the fifth in one case (5 per cent) and in the sixth in two cases (11 per cent).

It will be noted that in about 40 per cent of the cases the trouble developed during the first decade of life and that in more than 50 per cent the onset was before the age of 20. This is, of course, to be expected, since excessive trauma is more likely to be inflicted during operations on the tonsils and adenoids in children; also, the degenerative changes incident to hereditary syphilis are more active, and the acute infectious diseases are more common during this period of life. The youngest patient in the group was 7 years, and the oldest, 58. Twelve of the patients in the series were males, and five were females.

Fig. 2.—Same case as that shown in figure 1; after treatment.

The duration of the stenosis at the time of examination in the clinic varied from a few months to seventeen years. The shortest period during which symptoms had been noticed was four months. This was the case of a man, aged 52, whose tonsils had been removed because they were believed to be foci of infection. The faucial pillars, on both sides, had been destroyed, and the tense, soft palate was almost in contact with the posterior pharyngeal wall. The longest period over which symptoms had persisted was seventeen years, in a case of hereditary syphilis. There was marked variation in the size of the nasopharyngeal opening at the time of examination.

In one case the opening was approximately 1.5 cm. in width, but was so small in its anteroposterior dimension as to amount to almost

entire obstruction. In four cases of the group, there was complete atresia, even the small sinus, usually present, being absent. This is not in accord with the experience of most writers on the subject, who state that complete atresia of the nasopharynx is extremely rare. In two of the cases reported, the complete atresia was due to syphilis; in one case, to diphtheria, and in another, the cause was indeterminate. When an opening into the nasopharynx remained, it was usually in the median line and directly back of the uvula, if this had not been destroyed. At times, however, it was situated laterally. The thickness and density of the scar varied greatly in different cases. The nasopharyngeal stenosis seen in cases of scleroma is always concentric and appears to be due as much to infiltration and thickening of the wall of the nasopharynx as to cicatricial contraction.

Fig. 3.—Cicatricial stenosis of the nasopharynx following removal of the tonsils and adenoids; before and after treatment.

The symptoms of nasopharyngeal stenosis are dependent on the degree of obstruction. Often the patient accommodates himself to breathing through a very small opening, and may present practically no symptoms, even in the presence of marked contraction. With complete or almost complete atresia, the symptoms are primarily those of nasal obstruction. The lack of ventilation in the nose, together with blocked drainage, produces changes in the nasal mucous membrane, and infection of the accessory sinuses or deafness may follow. The sense of smell is interfered with, and if the condition has come on early in life, developmental changes, as regards both the facies and the contour of the nasopharynx, and possibly of the nasal fossae, may result. The voice loses its normal resonance, and irritation of the pharynx and lower respiratory passages may follow continuous breathing through the mouth.

The great variety of procedures that have been advocated for relief from cicatricial stenosis of the nasopharynx bears evidence of the difficulty of dealing with this condition. The most obvious method of treatment, and without doubt the one resorted to most commonly in the past, is incision of the cicatricial diaphragm and subsequent dilation. Yet, almost invariably this procedure fails. The opening made in this way usually contracts to such a marked extent that it becomes useless for respiration once the dilation is discontinued and often in spite of its continuance. On the contrary, in the cases of congenital stenosis this

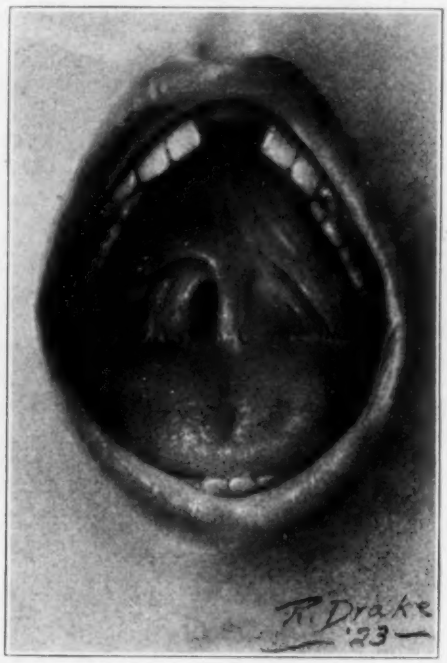

Fig. 4.—Stenosis of the nasopharynx due to hereditary syphilis.

method of treatment has proved entirely successful. Incision and subsequent cauterization of the raw surfaces with the galvanic cautery or with some type of chemical cautery are, likewise, not rational procedures. The wearing of a prosthetic appliance, held in place by dental bands, after reestablishing communication between the oral pharynx and nasopharynx, has been tried. Isaacs [6] reported a case in which this procedure was carried out; he did not mention the end-result. In the clinic, such an appliance was used in one case to hold a skin graft in place after the scarred attachment of the soft palate had been dissected

6. Isaacs, H. E.: Stenosis of the Naso-Pharynx Operation with Prosthesis, Laryngoscope **27**:885, 1917.

from the posterior pharyngeal wall. The graft did not take, however, and stenosis recurred. One might readily anticipate such a result, since the rigid dental appliance held the skin graft in contact with a surface that necessarily moved with each act of deglutition. The same situation must hold, even when epithelialization is complete, and the irritation produced by such an appliance would, in all probability, lead to failure. Diathermy has been used in the treatment for nasopharyngeal stenosis, with some success. The explanation of this, apparently, is the fact that the scar, following surgical diathermy, is often less dense, and has less tendency to contract, than that following most cutting operations or inflammatory conditions. This has been noted in the treatment for keloids about the neck.

Numerous plastic operations have been contrived for relief from this condition. In some of these, flaps of mucous membrane from the adjacent cheeks, soft palate and pharyngeal wall are utilized. In others,

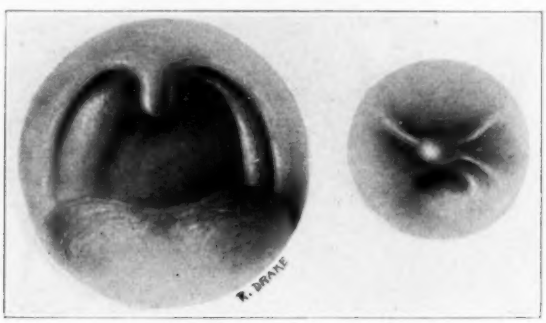

Fig. 5.—Congenital stenosis of the nasopharynx.

large skin flaps are introduced through a pharyngotomy opening. Curtis [7] reported a case of dense cicatricial stricture of the pharynx, due to syphilis, which was taken care of satisfactorily with a skin flap introduced through a suprahyoid pharyngotomy opening. Mackenty recently described an operation for relief from cicatricial stenosis of the nasopharynx that he has used successfully in several cases. His procedure consists in turning up two flaps of mucous membrane from the posterior wall of the pharynx, one on either side of the stenosed pharyngeal opening. Although the tissue comprising these flaps is taken from the pharyngeal wall, the base of each is situated at, and formed by, the posterior border of the soft palate. These flaps are doubled over onto the denuded superior or posterior surface of the soft palate, and the operation is completed by suturing them in place.

7. Curtis, B. F.: Cicatricial Stricture of Pharynx Cured by Plastic Operation, Ann. Surg. **33**:152, 1901.

Perhaps the simplest, and in most cases the most uniformly successful, procedure for correcting this condition, is that presented by Nichols.[8] He concluded that the reason the opening made by incision of the scarred membrane invariably contracted was the fact that healing always started at the apexes of this wound and progressed toward the median line. Drawing an analogy from cases of syndactylism, treated by inserting a seton at the base of the web until cicatrization took place, and then incising to this point, he inserted a silk suture through the small nasopharyngeal opening, well into the lateral extent of the region of scarring. This suture was tied and left in place until a cicatrized tract developed. The posterior border of the soft palate was then freed

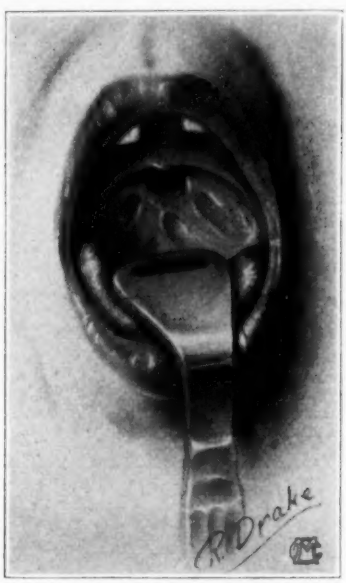

Fig. 6.—Stenosis of the nasopharynx due to rhinoscleroma.

from its attachment to the pharyngeal wall out to this point. In 1896, before the American Laryngological Association, he[8] reported a group of thirteen patients treated successfully by this method.

In the last six cases of cicatricial stenosis of the nasopharynx seen in the Mayo Clinic, essentially the same procedure as that described by Nichols has been employed with uniform success. The variation has consisted merely in adding a small lead weight, which is clamped on over the ends of the suture, and allowing the suture to cut through of itself. This usually requires from one to two weeks. The suture and weight are almost invariably swallowed. The possibility of aspiration

8. Nichols, J. E. H.: The Sequelae of Syphilis in the Pharynx and Their Treatment, Tr. Am. Laryngol. Ass. **18**:161, 1896.

has been considered but, thus far, difficulty has not been experienced in this regard. After the suture cuts through, it is replaced by another, taking a wider lateral bite. Subsequent dilation with rubber-tipped Kelly forceps has been employed in most cases, regardless of the primary method of treatment. In a few cases, a soft rubber tube with a self-retaining cuff at either end was inserted for short periods, with marked benefit.

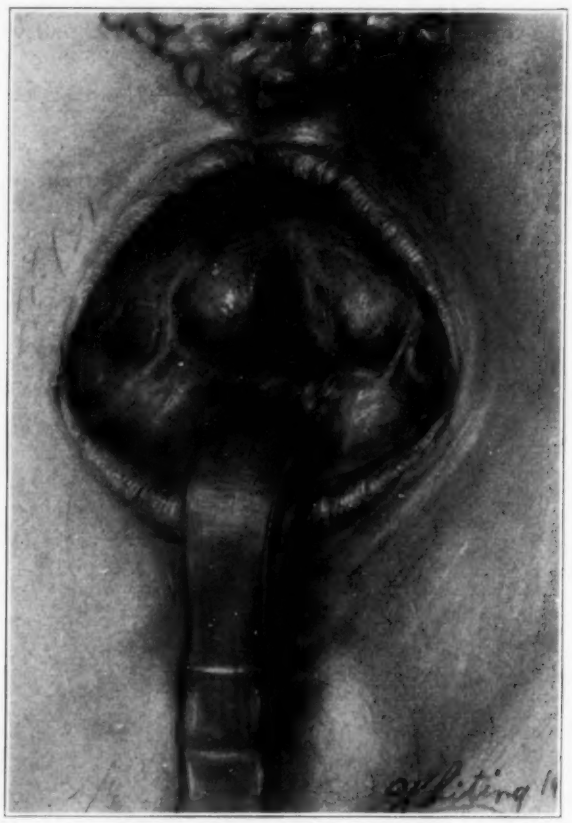

Fig. 7.—Stenosis of the nasopharynx due to an indeterminate type of granuloma simulating rhinoscleroma.

In the clinic, prior to 1922, patients were treated by various other methods, and the results were, as a rule, less satisfactory. Forcible dilation, under anesthesia, with repeated use of a hard rubber dilator, was employed in one case. The stenosis recurred. In another case, a flap of mucous membrane from the cheek was turned in along the posterior border of the denuded palate, after freeing the region from scarring and attaching a dental appliance to the molar teeth on the opposite side, to prevent biting of the pedicle. In still another case,

a soft rubber tube of large caliber, with well rounded ends, was introduced through a transverse incision in the thick cicatricial diaphragm that was producing complete atresia. This was held in place by means of silk cord passed through either nostril and tied beneath the collumella. It was worn continuously for several weeks after the margins of the incision had epithelialized. A satisfactory opening resulted, and this had changed little in size at the time of observation four and a half years later. The patient did not wish to have it enlarged for fear of regurgitating fluids through it, as had occurred immediately following removal of the large rubber tube. Canuyt [9] described a method similar to this, except that a gauze pack was kept in the nasopharynx for twelve days following the operation, and then replaced with a rubber tube. Soft rubber tubes also have been employed at the clinic in the treatment for cicatricial stenosis of the nostrils and of the external auditory canals in a number of cases, and have yielded excellent results. These soft tubes have proved more effective than hard rubber dilators. They produce much less irritation and apparently bring about absorption of a certain amount of scar tissue. In the cases of nasopharyngeal stenosis incident to rhinoscleroma, treatment was directed entirely toward controlling the infection. This consisted of the application of radium and surgical diathermy locally, and potassium iodide internally. As the infection cleared, the stenosis disappeared to a great extent, so that further treatment for the contracted nasopharynx was not necessary.

Four of the eighteen patients with nasopharyngeal stenosis who have come under observation at the Mayo Clinic did not stay for treatment; two, treated respectively with dilation and by skin graft, did not receive permanent benefit. One patient, with coincident esophageal stenosis, has, as yet, not received treatment for the nasopharyngeal stricture. One patient, in whose case a flap was taken from the buccal surface of the cheek, was partially relieved; the nasopharyngeal opening was enlarged from a diameter of 2 to 8 mm. One patient, treated by incision of the diaphragm and the wearing of a rubber dilator, was relieved to such an extent that, as previously stated, he did not wish the opening made larger. The nasopharyngeal aperture measured 5 by 12 mm., whereas complete atresia had been present. Six patients treated with weighted sutures received complete relief from their symptoms. One of these had come with complete atresia, and two others with openings from 2 to 3 mm. in diameter. Two patients with rhinoscleroma were relieved from nasopharyngeal stenosis, as was also one other patient with a granuloma of apparently similar nature.

9. Canuyt, G.: Les stenoses cicatricielles du naso-pharynx, Rev. de laryngol. **43**:23, 1922.

COMMENT

There has been a change in tone of the reports on stenosis of the nasopharynx from the earlier writings on the subject, which are in general extremely pessimistic, to those of more recent date. Labit [10] said: "It must be frankly acknowledged that no means has yet been found which will afford permanent relief." Mackenzie, Scheck, Cartaz, Lichtwitz and Hajek [11] also have written in much the same vein. Ballenger,[12] likewise, noted that treatment for syphilitic cases results in failure in the majority of cases. On the other hand, the reports of Nichols and Mackenty are outstanding in their optimism. The prognosis in such cases in the Mayo Clinic has likewise passed from one of decided pessimism prior to the use of the seton to one reasonably optimistic since its introduction. A favorable prognosis is now given as a routine, provided the patient is able to remain under observation for a sufficient length of time. This period varies greatly, depending on the cause of the condition, the degree of stenosis and the thickness and density of the scar tissue present. The shortest period any of these patients has been under observation for treatment was five and a half weeks, in a case of complete syphilitic atresia. The longest period was eight months, in the first patient treated with the seton at the clinic. The average period of observation and treatment, in the more recent cases, has been a little over three months. The stenosis due to rhinoscleroma has been slower in responding to treatment, requiring from six to eight months.

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10. Labit, quoted by Nichols: Tr. Am. Laryngol. Ass. **18**:161, 1896.

11. Mackenzie, Scheck, Cartaz, Lichtwitz and Hajek, quoted by Nichols: Tr. Am. Laryngol. Ass. **18**:161, 1896.

12. Ballenger, W. L.: Malformations of the Pharynx: Stenosis of the Pharynx, in: Diseases of the Nose, Throat and Ear, ed. 5, New York, Lea & Febiger, 1925, p. 370.

THE OPERATIVE TREATMENT OF CLEFT PALATE

A NEW METHOD *

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It may be of interest to take up in short review the past history of surgical procedures for the cure of cleft palate, especially between the years 1760 and 1875.

A careful search of the literature, so far as available in the New York Academy of Medicine Library, gave meager returns prior to the year 1760. Sprengel,[1] in his "Geschichte der Chirurgie," mentioned a Dutch surgeon, H. van Roonhuyse (1625) as describing the cleft palate in his "Genees- in Heelkonstige Aanmerkingen," but this book could not be located. Sudhoff,[2] in his monographs on medieval surgery in the "Studien zur Geschichte der Medizin," gave illustrations of instruments for use in oral surgery.

During the second century, Galen called attention to this deformity but did not go beyond making a few casual observations and naming it Ladocheilos (lip like a hare). Harelip is mentioned by many other authors (Celsus, Fabricius d'Acquapendente—1537-1619, de la Vauguion).

It was during the period between 1760 and 1875 that most of the fundamental principles were worked out, with much labor and infliction of pain, and as usual this unfolding of knowledge was incredibly slow, even the most obvious principles often being overlooked. As an example of this, for twenty years after the discovery of anesthesia this inestimable solace was denied these sufferers.

Malgaigne,[3] in his edition of the "Complete Works of Paré," mentioned Paré as the inventor of an artificial palate for the improvement of speech, but stated in a footnote that it is only in the 1579 edition of his works that he claimed to have invented it. In an earlier edition of 1561, he merely described this device and did not claim credit for the invention.

The first attempt to cure cleft palate by operation appears to have been made by a French dentist, Le Monier, about 1760. Though that experiment was subsequently repeated in two or three instances, staphylorrhaphy (the correction of defects of the soft palate) did not become an established operation until after Roux' famous case in 1819.

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* Submitted for publication, June 4, 1929.

1. Sprengel: Geschichte der Chirurgie.
2. Sudhoff: Studien zur Geschichte der Medizin, nos. 10 and 11.
3. Malgaigne, J. F.: Ambroise Paré, Paris, Reillière, 1840-1841, vol. 2, p. 607.

The improvement of speech after Roux' operation was so wonderful that the friends of the patient recognized him only with difficulty as the same person. Though the operation was often repeated, it was by no means invariably successful. Velpeau, writing in 1839, referred to a young girl who suffered the operation five times without benefit, and he mentioned several fatal cases in Roux' own practice.

In 1827, Stevens,[4] an American, reported a cure by the technic devised by Roux of Paris, this being the first attempt made in America. The patient was placed in the sitting posture. With a rolled handkerchief for a mouth gag, a flap was raised from both sides of the cleft, the stitches being inserted previously. Only three were used. "The patient went to bed to prevent hunger and did not speak or swallow anything (not even his saliva) for four days. At the end of the fourth day the threads were removed and the parts were perfectly united." The extent of the cleft is not stated. It must have been only a cleft of the soft palate. He related in his paper that the operation was first performed by Roux of Paris on a Mr. Stephens of Canada with complete success.

Alcock of London operated on one patient five times only to meet failure due, Stevens believed, to the surgeon using a granny instead of a surgeon's knot. He palliated this criticism by stating that Alcock recognized the imperative need of speech instruction following the operation and the advantage of a loose "velum pendulum palati in order to close the opening of the posterior nares."

In a paper written by Fergusson,[5] 1845, he gave Alcock the credit for the first operation done in England in 1821. He further stated: "More than two years ago the author of the operation, Roux, had performed the operation more than one hundred times with a most satisfactory amount of success, two-thirds of the simple cases and one-third of those which were complicated having derived benefit from the proceeding."

In 1843, Mutter of Philadelphia claimed cures in eighteen of twenty cases.

Prior to 1843, it is related that "J. Mason Warren (Boston) had treated fourteen cases, in thirteen of which he had attained most signal advantage by his judicious attempts to close the fissure by the operation in question."

It seems probable from a perusal of such literature as can be obtained that up to about this period the attempts were made entirely on the simple forms of cleft palate, probably clefts of the soft palate.

4. Stevens, A. H.: Staphylographe or Palate Suture Successfully Performed, N. Am. M. & S. J. **3**:233, 1827.

5. Fergusson, W.: Observations on Cleft Palate and on Staphylorrhaphy, Tr. Med. Chir. **28**:273, 1845.

The defect of the hard palate was closed by an obturator. Warren seemed to have been the first to show that clefts of the hard palate could be closed by dissecting the soft tissues from the bone on each side of the fissure. "Carrying his knife towards the alveoli to such an extent as to make a flap sufficiently broad to join its fellow on the mesial line," he pared the edges of the cleft in the soft palate and stitched the flaps together much in the same manner as is done now. He was criticized for denuding the bones of the hard palate.

Fergusson, in a lengthy description of Warren's technic, brought out an interesting point: "Taking the vault of the hard palate in its natural condition as being nearly semicircular, it is evident that by bringing down the soft parts on each side towards the tongue ample flaps may be obtained so that there would be little or no dragging by the stitches." In discussing modifications of the technic he stated: "Extra incisions have been made by some with the intention of facilitating the approximation of the edges of the fissure."

Dieffenbach,[6] in 1834, was the first to propose a longitudinal division of the flaps between the alveoli and the stitches for the relief from tension. This was approved and used by Pancoast[7] of Philadelphia, who added to it the dividing of the insertions of the palati muscles "so as to prevent them straining the sutured edges of the palate asunder."

Roux adopted these various modifications of his original technic and added another most important step to the procedure: "When the gap is wide, especially when the hard palate is defective, by transverse incisions, he separates the soft palate on each side from the posterior margin of the palati bones which permits the more ready approximation of the movable parts towards the mesial line."

Fergusson added the division of the posterior pillar to this technic.

Assembling the facts just related, one has all the salient principles of the operation for cleft palate outlined and put into practice during the first half, or less, of the nineteenth century.

Langenbeck's name has been intimately associated with the foregoing principles, and it is generally believed that he merits the credit of discovering them. The literature does not carry this out, as is frequently the case in the evolution of a surgical procedure. Knowledge, coming in driblets and from many sources, is correlated by a person who wrongly gets all the credit of the discovery. The pioneers and their labors are too often forgotten.

It is to the credit of the courage of these pioneers that up to about 1850 and even for a long time after the discovery of anesthesia, the operation for cleft palate was done without the aid of anesthesia. Thus,

6. Dieffenbach: Lancet **1**:695, 1834-1835.

7. Pancoast: Am. J. M. Sc., vol. 32, p. 171.

children were excluded, as the patient had to be old enough to cooperate. Unspeakable misery of course accompanied the procedure. First the operation was performed, which required from one to two hours; then four days of starvation followed, when neither food nor water, not even saliva could be swallowed.

In 1879, Ashhurst [8] stated: "Until a few years ago it was an accepted dictum that no interference should be recommended until the patient had acquired sufficient age to earnestly desire relief and to cooperate with the surgeon in the operation and after-treatment." He emphasized the horrors of the operation in the preanesthetic days. He related that as late as 1867, "J. M. Warren, who had a larger experience in the treatment of cleft palate than any other American surgeon, had operated but once on a young child and expressed the opinion that it was generally necessary to wait until the patient was old enough to fully appreciate the importance of the treatment and to submit patiently to pain and inconvenience." Warren's own statement was: "This was one of the very few operations in which the use of anaesthesia was inadvisable."

The same opinions were held by Fergusson, who later recanted and became convinced that children might be operated on and anesthesia given. It was about this period that the advantages of operating during early life, especially in relation to speech, began to dawn on the surgeon. The age of 3 to 4 was set as the most suitable time.

Up to this time the uplifted flaps did not include the periosteum. Langenbeck then made his inestimable contribution by including the periosteum in the flaps, claiming that it contained the osteogenic elements for the formation of a new hard palate. Ever since then the various operations are spoken of as being modifications of Langenbeck's method. Ashhurst, however, gave most of the credit to Fergusson and to Warren. Ashhurst (1879) strongly advocated early operation (third and fourth year) and the use of anesthesia.

This concludes what might be called the early history, or formative period, of the operation, in which the fundamental principles were fairly well worked out. Since the beginning of the present century a mass of literature has grown up around this subject too extensive to be more than glanced at in this paper.

Many unnecessary and surgically unsound procedures have been advocated and tried, only to fall into deserved oblivion. Such, for instance, was McKernon's method of tracheotomy, and the packing, during healing, of the nasal and pharyngeal spaces. This was a reflection of the antiseptic period in surgery when as little as possible was left to nature. As late as 1907, Makuen [9] championed this method. He

8. Ashhurst, J.: Cleft Palate and Its Treatment, M. Rec. **16**:145 (Aug. 16) 1879.

9. Makuen, G. H.: Operations for Cleft Palate and Their Results; Especially in Respect to the Improvement of Speech, New York M. J., July 27, 1907.

admitted that "in the technique of the cleft palate operation there is yet much to be desired although it has had ample time for improvement." He regretted that the operation is in the hands of the general surgeon —"The worst kind of bungling has been done in this delicate operation by men who report successful appendiceal operations by the thousands." He warned the overenthusiastic that they should withhold their hand in cases in which operation would evidently prove useless. Mauken's conclusions are sound and stand for all time:

1. The operation should be advocated only when there is a fair likelihood of success. 2. It should be done only by those possessing skill and experience. 3. If multiple operations are probable, the patient, or parent, should be so informed. 4. The operation should be done as early as possible. 5. The operation aims at the improvement of the patient's general health, his general morale and his speech. 6. Speech education is imperative.

Lane's [10] flap-turning operation is too well known to require a detailed description here. It is a most ingenious method, especially in wide clefts, of closing off the nasal from the oral cavities. Were that the only object desired, it would be an admirable procedure, but it seems to me that palatal function is better conserved by some modification of the so-called "Langenbeck Method." It is the opinion of many writers with large experience in this work that Lane's operation is inimical to improved speech. It is my opinion that this operation has a limited and fixed place.

Lane has proved the feasibility of operating on young infants. He has great personal skill in executing his operation quickly and with a minimum loss of blood; yet he reported a mortality of 9 per cent from bronchopneumonia and gastro-enteritis and too low a percentage of cures.

Stahl,[11] in 1923, reported from the Charité clinic (Berlin) sixty-two operations, with a complete closure in 58.7 per cent, partial closure in 25.6 per cent and failure in the remainder. He stated the belief that the operation should be deferred to the fourth, fifth and sixth year.

Veau [12] reported 102 operations, with two deaths, seventy closures by one operation and ninety-one by one or more operations.

Of the many recent papers published both here and abroad, the most complete and rational is by Rayner [13] (England). He performed the opera-

10. Lane, W. A.: Cleft Palate and Hare Lip, ed. 3, London, Adlard & Son, 1916, p. 102.

11. Stahl, O.: Operation for Congenital Cleft Palate: End-Results of Mano-staphyloplasty by Langenbeck's Method, Arch. f. klin. Chir. **123**:271 (March 15) 1923.

12. Veau, V.: The Anatomic Results in 102 Staphylorrhaphies, Bull. et mém. Soc. nat. de chir. **50**:497 (April 12) 1924.

13. Rayner, H. H.: The Operative Treatment of Cleft Palate, Lancet **1**:816 (April 18) 1925.

tion on one hundred and twenty-five patients over a period of fourteen years. Of these he traced sixty-six. Twenty-one were partial failures, mostly in the hard palate. In some of them the speech was materially improved by an obturator. The patients in the cases of total failure (7 per cent) were materially worse off than they would have been had no cure been attempted. The operation was rarely done after the second birthday; operations on clefts of the soft palate were done at an earlier age, depending on the type of defect and the physical condition of the child. He warned against extensive division of the elevator muscles of the palate unless closure cannot be otherwise obtained. It was in cases in which extensive division was carried out that the most pronounced defects in speech resulted, in spite of a good appearance of the palate.

He attributed the failures to the following causes: operating on unsuitable subject, chronic sinus disease, latent nasal infections following scarlet fever, measles, etc., too extensive lateral incisions causing ischemia of the flaps, tying of sutures too tightly, inversion of epithelial edges, especially in the hard palate—flap tension coming on two or three days after operation though the flaps may be flaccid at time of operation.

Of sixty-seven cases of complete clefts, forty-two resulted in complete union, three after two operations (62 per cent); partial union resulted in twenty-one cases and failure in four. In the incomplete clefts he secured 91 per cent cures. He had one death in the whole series (0.7 per cent) from secondary hemorrhage four hours after operation.

Addison [14] expressed the opinion that failures in operations on a cleft palate are due to tension and sepsis. He stated the belief that inadequate lateral incisions account for the former, and diseased tonsils and adenoids, infected teeth and gums and general debility account for the latter. To relieve tension he makes long lateral incisions. Beginning on the lower jaw on either side, the incision is carried up the ramus of the jaw onto the upper jaw to the last molar tooth, then forward in close contact with the teeth, as far as seems necessary. He claimed that these incisions are outside the tensor and levator palati muscles. He made no mention, however, of the posterior palatine arteries and nerves. The tonsils and adenoids should be removed as a routine practice at least four weeks before the operation on the palate. This author is against all forms of tension sutures. He prefers the Langenbeck, with mediam suture, to the Lane operation.

Ladd,[15] 1926, condemned the Lane operation on account of its negative functional results. His choice of time for the palate operation

14. Addison, O. L.: Cleft Palate: Some Pitfalls in Operation, Lancet **1**:818 (April 18) 1925.

15. Ladd, W. E.: Harelip and Cleft Palate, Boston M. & S. J. **184**:1016 (June 3) 1926.

is from eighteen months to two years. Longer delay, he claimed, widens the cleft in the soft palate. Lateral incisions are used, care being taken to avoid the palatine artery. His mortality is 3 per cent, occurring in patients under 2 years of age. Forty-five operations done on patients after the age of 2 years were attended by no mortality. In 88 per cent the results were good; in 7.5 per cent of these cases the patients required more than one operation. Ladd's paper is based on 400 cases of harelip and cleft palate.

TECHNIC OF OPERATION

The following is not a discussion of the whole subject of cleft palate, but a brief description of the technic devised and employed by me for the closure of the cleft in the hard and soft palate. All procedures, such as plastic bone work on infants, leading up to the final operation and the methods employed by other operators are omitted.

My purpose is to present my own conclusions drawn from nearly 200 operations and to describe a method which after much experimentation I have finally evolved and which in the hands of others and in my own has enormously increased the percentage of cures.

Part of the technic was in operation for several years prior to 1914 when, in a candidate's thesis presented before the American Triological Association, I gave a detailed description of it, with an analysis of sixty-four cases, traced in a series of ninety-two. The completed technic to be described here took its present form in 1915.[16]

The failures to improve the speech far exceed the operative failures. I am convinced that this must so remain unless the patients can be brought to operation before the end of the second year and this followed by prolonged postoperative speech instruction. The earlier the operation, given a good result, the better is the outlook for normal speech. Mutilating operations which interfere with the musulature or enervation of the soft palate invariably give defective speech. If the child is healthy, the cleft should be closed between the twelfth and fifteenth month of life. The closure of the lip and (in complete clefts) the replacement of the premaxilla should be done as soon after birth as the condition of the child will allow. No one operative method can be depended on for all cases.

In a few wide clefts with low arch, in some secondary cases, in which all hope of improved speech is abandoned, Lane's method serves as no other does in closing the oral from the nasal cavities. This closure adds at least to the patient's comfort.

Too little attention is paid to the preparation of the child with cleft palate and harelip for the two surgical ordeals which he must

16. MacKenty, John Edmund: Ann. Otol. Rhin. & Laryng., March, 1915.

face. To submit to surgical procedures a weak, undernourished anemic child is courting disaster. In infants the amount of milk taken at each feeding should be ascertained exactly by weighing the child before and after nursing; spoon feeding and even tube feeding may have to be employed to ward off starvation. Nursing should be encouraged from birth, even if no milk is extracted, as the secretion of the salivary glands is thereby stimulated. The act of nursing is practiced for future use, and normal lactation is stimulated. From four to eight weeks after birth, the premaxilla may be replaced and the lip closed. In robust children, the palate may be operated on a few months later; but it is generally better to wait until the end of the first or the beginning of the second year.

In older children the tonsils and adenoids should be removed at least two months prior to the attempt on the palate. Even if the tonsils look normal, they are often a menace on account of the infective material in their crypts. A traumatic tonsillectomy with subsequent scarring and contraction lessens the hope of success by still further widening the soft palate cleft. Chronic nasal sinus disease is a contra-indication to operation. Diseased teeth and gums may require attention. It must occur to every one that early operation, even if the risk incurred is a little greater, has many advantages. The absence of teeth allows a freer flow of blood over the alveoli into the flaps; sinus, tonsillar and adenoid diseases are rare in patients under the age of 15 months. Teeth, even when healthy, harbor infection. Congenital syphilis should be carefully excluded.

I do not approve of procedures aiming at the bending of the alveoli toward the midline by wires passed through the upper jaw. A thorough trial of this much advocated method of lessening the width of the cleft has convinced me that it is mechanically defective and too often fatal in its result. The presence of the cleft is due, in my opinion, not only to spreading of the alveoli but to a considerable extent to lack of development of the palatine plates of the maxillary and palate bones. What strikes one in examining the skull of an infant is the short distance between the alveolar processes of the upper jaw and the base plate of the cranium; any effort to displace these processes inward must often result in their rotation and in a warping of the cranial floor. The former may result in faulty dental eruption, and the latter may produce death.

It has been customary in replacing the premaxilla (in double cleft palate) to resect a v-shaped piece from the septum. When possible, sufficient resection of the septum and dislocation of the premaxilla should be carried out to allow the replacement with as little rotation as possible. The teeth then erupt in more correct alinement.

In single cleft palate the long arm usually holds the premaxilla (central cleft with division of the premaxilla is rare). A chisel is passed through the bone between the two tooth buds if possible, and the projection is fractured into position and there wired. I have never been able to bend this bone into position and hold it there without previous fracture.

To present this procedure more intelligently, I shall lead up to it by a brief summary of the mechanical principles and handicaps involved.

The adverse forces present in this surgical field are so many and so constant that one wonders why failure is not the rule. Chief among these forces are septic infection and traumatism.

SEPTIC INFECTION AND TRAUMATISM

The aim is to secure primary union in a septic field (a surgical paradox). Surgeons were slow to realize in operating in an unclean field that rigid asepsis was imperative. Hope of success rests on a belief that the patient is more or less immune to his own strain of mouth bacteria and on a technic which rigidly excludes other strains and on a minimum amount of traumatism.

Traumatism may be brought about in the following ways: injury to the tissues by rough handling during operation; wounding of the palatine arteries, the chief source of blood supply to the flaps; tongue pressure, present at all times and particularly during deglutition; eccentric pull on the stitches during deglutition; tension on stitches due to failure on the part of the surgeon to bring the flaps into easy opposition and contact of stomach contents with the unclosed wound, due to vomiting during operation.

It is a common observation that infections in wounds and postoperative complications are more common during the winter and summer. This applies in a marked degree to this particular operation and to all operations on young children. In my experience the percentage of healing was cut in half during these unfavorable periods. Hence for years I have advised that this operation be done only during the spring and fall.

The technic followed in the operation is the logical application of the foregoing principles.

As the operation, even in the most expert hands, is not a short one, depression from excessive anesthesia should be carefully avoided. Many failures may be traced to this cause since it acts not only immediately as a tissue depressant but remotely in stopping food absorption (a serious condition in children). Postoperative acidosis is fatal to union. The strictest asepsis is observed. The face and the entire mouth are painted with a 2 per cent iodine, and a 2 per cent acriviolet

is dropped into the nose. Gloves and masks are worn by all assistants and by the anesthetist.

The patient's head rests on the operator's knees over the head of the table. This position gives absolute control of the head and affords the best possible view of the operative field. One assistant stands on each side. Suction is used to take the place of sponging as it causes much less traumatism. Sponges are used for compression and for wiping the edges of the flaps before tying the stitches.

With a right angle knife, an incision is made along the edge of the cleft on both sides. Brophy elevators are used to lift the soft tissues, including the periosteum, away from the bone of the palatal processes. This separation proceeds from the center outward toward the alveolar

Fig. 1.—Right angle knife making incision through mucous membrane and periosteum along the edge of the cleft from the posterior end of the hard palate on one side around the anterior angle to the same point on the opposite side.

process. When no teeth are present, the flaps may be lifted away to a point level with the apex of the alveolar ridge. In older patients, when teeth have erupted, too close an approach to the teeth may seriously damage the blood supply. The posterior palatine artery must not be injured. The periosteal reflection around the posterior end of the palatal process is cut outward, thus allowing the palate to drop into a horizontal position. Nicking of the posterior pillars is sometimes indicated in order to make this flattening of the palate complete. This is essentially the Langenbeck method (Warren-Fergusson-Langenbeck).

The edges of the soft palate are denuded, making a raw edge as wide as possible with the removal of the least amount of tissue. Many

methods of placing the stitches are advocated. I prefer the straight through and through stitch of fine silkworm gut for the soft palate and the end on mattress stitch of Equisetene for the hard palate (see fig. 15). This mattress stitch everts the edges of the thin flap which lie over the hard palate and makes union more probable by increasing the area of the coaptated surfaces. At this point my method diverges from the others.

In 1914, I published a paper on this subject giving the smaller points on technic. Here the question of tension was discussed in detail. Out of the experience gleaned from nearly 100 operations done prior to that time two principles emerged. One dealt with the

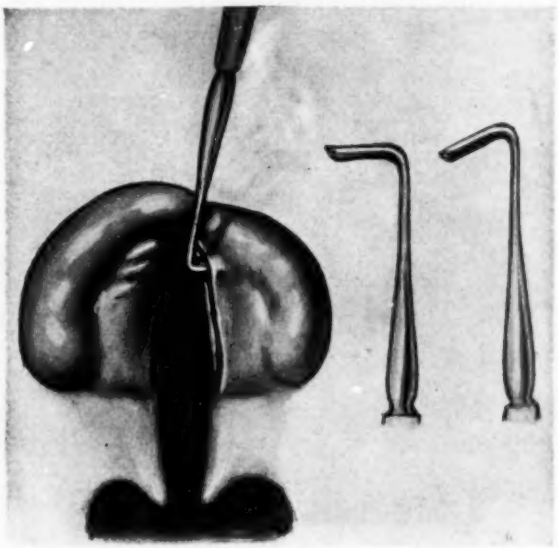

Fig. 2.—Elevators of different angles lifting the periosteum and mucous membrane away from the bone. This separation is carried outward to the alveolar process when teeth are present, and up to the apex of the process when teeth are absent.

relief from tongue pressure and the other with lateral tension on the flaps. The following is quoted:

Tongue pressure as a factor in producing strain, so far as I am aware, has been overlooked. That it is a potent cause of failure needs no proof when we consider the amount of upward force exerted by the tongue in each act of swallowing and that this act takes place every few minutes more or less unconsciously and quite independently of taking food.

For several years I have been using a device for the relief of this strain and now feel justified, on account of the good service it has rendered me, in presenting it for trial by others. A plaster cast of the palate is taken and on this cast is made an obturator which fits accurately along the inner margin of the gingival

ridge in infants and along the base of the teeth in older children. The obturator is made of platinoid wire, heavy at the rim and light inside. The obturator is cross barred, the wires being soldered at the points of crossing. After the completion of the operation the obturator is sewed to the gums of infants and tied to the teeth in older children. Three points of attachment are sufficient—two well back on each side and one in the centre in front. The obturator is left in until the stitches are removed. It in no way obscures inspection of the wound or interferes with cleansing. I use it in all cases where the cleft extends beyond the soft palate. It should be made flat or curved slightly upward and should not project far enough back to impinge against the downward curve of the soft palate.

At that same period, 1914, I was endeavoring to relieve lateral flap tension by inserting small hook retractors into the lateral incisions or into puncture holes if lateral incisions did not seem indicated. The following is extracted from the aforementioned paper:

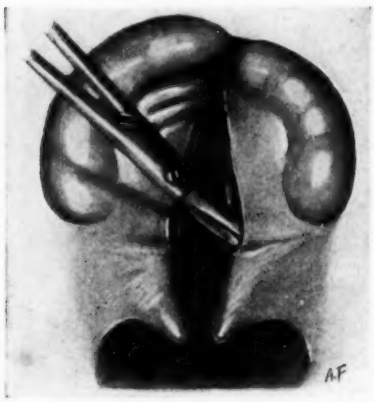

Figure 3

Figure 4

Fig. 3.—Incising the periosteum where it is reflected around the posterior end of the hard palate onto the floor of the nasal cavity. This is carried outward to the wall of the nasopharynx.

Fig. 4.—Soft palate dropped into horizontal position. When teeth are absent, a further drop and elongation of flaps may be obtained by carrying the separation to the top of the alveolar process.

Then I devised flat-faced small retractors of silver. These can be made any width desired. The arms are about equal in length, the object being to secure a good grasp on the inner edges of the lateral incisions or punctures. The arm on the palate or buccal side has an eye in its end to which a linen thread is secured. The threads of the two retractors are tied and drawn together sufficiently to take all tension off the central line. The retractor can be made for each case by the surgeon from virgin silver plate rigid enough to maintain its shape when bent. It should be of sufficient width to insure it against cutting into the edge of the lateral incision.

The obturator devised over twenty years ago has been retained in its original form, except for refinement of construction, and has been used ever since in all cases in which the cleft extends anterior to the hard palate. The hook retractors were not entirely satisfactory and were replaced by the lead or silver band now used (figs. 10, 11, 12

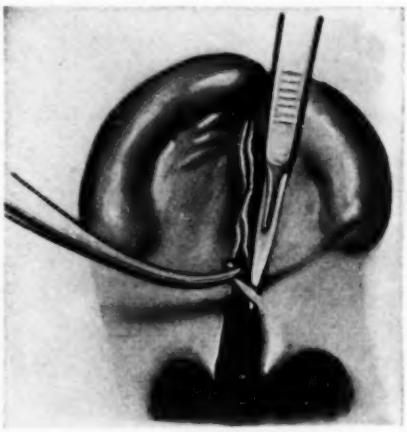

Fig. 5.—Denudation of edges of the soft palate. The knife points inward toward the median line in order to widen the denuded area.

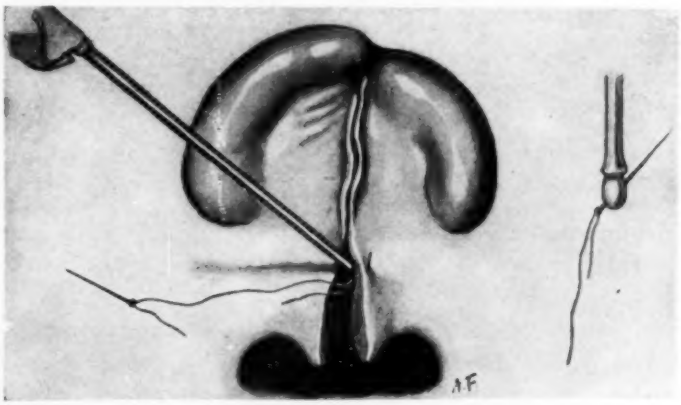

Fig. 6.—Method of using straight needle. It is hooked from above downward through the tissues, taking a liberal bite.

and 16). This consists of a strip of lead or silver slightly less than 1 cm. wide and 15 cm. long. One end of this band is cut to a point. At this point a small hole is punched for the attachment of a stout thread Two rows of holes 0.5 cm. apart in cross-section and the same in longitudinal section are punched along the extent of the middle third of the

band. A thread carrying a large curved needle is tied through the hole in the pointed end of the band (fig. 17).

This band has been used to relieve tension in two situations, one in front of the emergence of the palatine arteries and the other behind the posterior ends of the alveolar processes. When the stitches in the soft palate are all in place, the gap between the flap edges can be estimated (fig. 7). Then the lateral incisions which are to carry the band (or bands if two are used) are made (fig. 8). It is evident that the farther these incisions are made from the line of union, the better for the circulation along its edge. At a point on both sides just behind the alveolar process and in the angle where the buccal surface curves onto the soft palate, a knife is plunged through the tissues to emerge in the nasopharynx. The line of perforation is

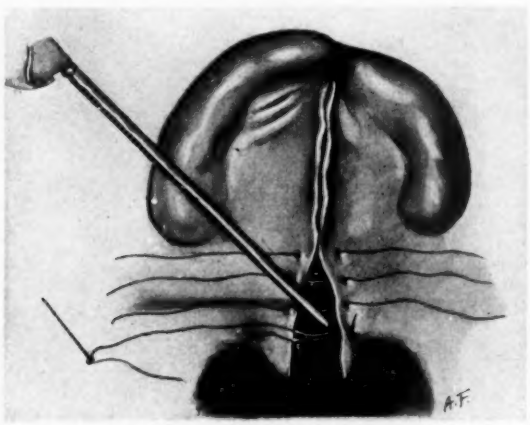

Fig. 7.—Four stitches in place. The last one must be well forward of the point where the soft palate curves onto the uvula in order not to strangulate the uvula. The bite of this stitch should be a little closer than the others to the raw edge for the same reason.

directed slightly to the center line. Through this hole an artery clamp is passed and gently opened, the object being to stretch the tissues (fig. 9). Slowly and gently the soft palate is worked over toward the center. It is sometimes necessary to extend the incision along the posterior end of the alveolar process. This incision must never curve forward around this end since the palatine artery is found at this point. The same procedure is carried out on both sides until the flaps meet with ease at the center line. The curved needle holding the band is now backed, eye first, through the stretched incision, carried across the nasopharynx and brought out on the buccal surface through the corresponding incision on the opposite side (fig. 10). The thread then leads the band through in the same way (fig. 11). The stitches are now tied in the soft palate (figs. 12, 13

and 14). More stretching of the tissues toward the center line may be required as each stitch is tied to secure coaptation without tension. The importance of complete relaxation cannot be too strongly stressed. In some cases the lateral incisions gape so wide on either side that the nasopharynx can be seen through the openings. If the center unites.

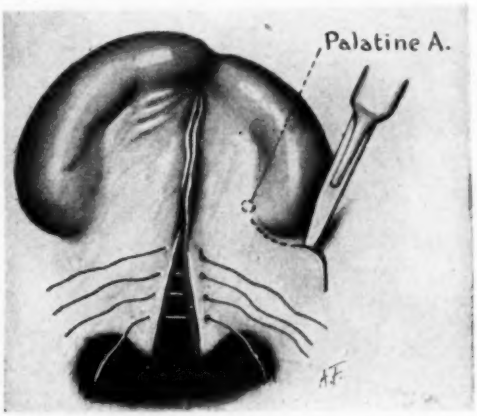

Fig. 8.—Method of making posterior lateral incision. Note that the knife enters the tissues with a distinct slant toward the center of the nasopharynx and at the buccal angle of the palate. The length of incision depends on the amount of tension present.

Fig. 9.—Stretching the tissues of palate toward the center line.

these large lateral holes always close promptly. The ends of the lead band are now crossed and tightened until all tension is overcome and the palate lies flaccid at the line of union. It is then secured by passing a thread through the holes in the band which come opposite each other. The overlap in the band is cut away just distal to the

point of tying and the ends are adjusted (fig. 15). This results in a band ring passing across the buccal surface of the soft palate through the lateral incisions and across the pharyngeal surface of the soft palate. If required, a similar band may be placed so as to relieve tension in the region of the hard palate, though this is rarely necessary. Here the

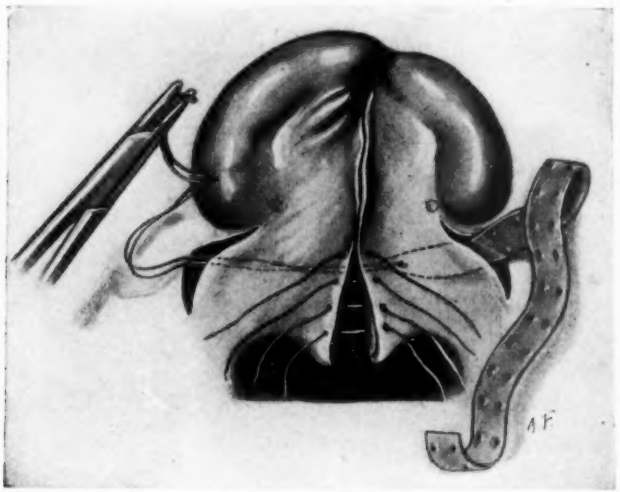

Fig. 10.—Method of conducting the band through the nasopharynx.

Fig. 11.—Band in position.

lateral incisions are made as far out as possible and the tissues stretched to the center as before described.

These bands have been employed since 1914 in all cases in which tension exists, and that is in the large majority. Upright or end on mattress sutures are placed in the hard palate flap (fig. 15). In figure 15, *A* represents the correct placement of the stitch, *B*, the incorrect. The uvula is closed according to figures 12, 13 and 14, only the mucous

membrane being pierced by the stitches. The obturator is now placed in position and secured by three threads, transfixing the alveolus as shown in figure 16. When teeth are present, clasps are used on the obturator at three points—one anteriorally and two laterally. These clasps fit the teeth and are secured by turning a screw which fits a band around the tooth.

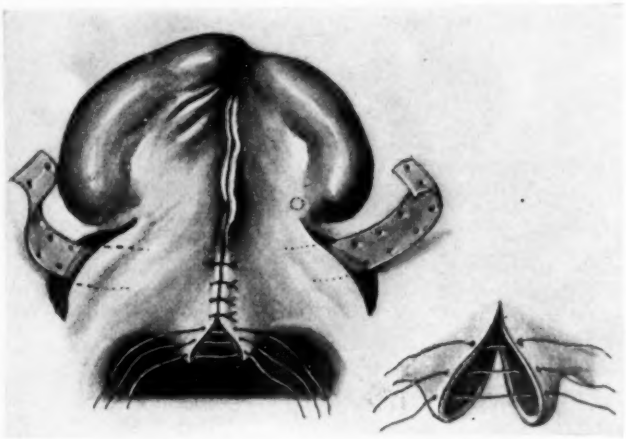

Fig. 12.—Tying of stitches and closing of uvula. Note that stiches in uvula pass only through the mucous membrane.

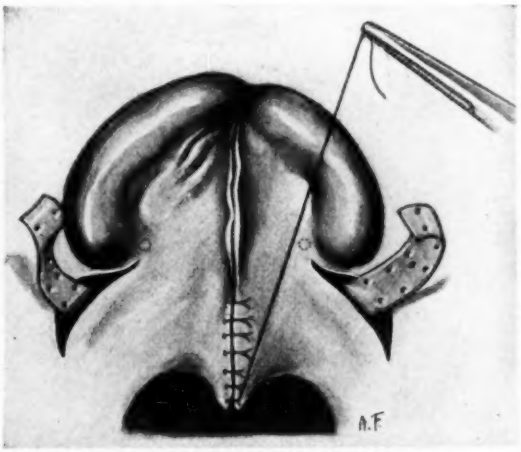

Fig. 13.—Last stitch in uvula retained as a tenaculum to evert the palate and expose the upper or posterior surface.

This method puts the palate completely at rest during the healing period. Tongue pressure is entirely obviated. The muscular contractions of the soft palate during crying and deglutition exert their force against the band and not against the line of union.

My percentage of primary healing has been advanced at least 20 per cent over those reported by me in 1914. The complete union in uncomplicated cases has been over 95 per cent. No prognosis as to union can be given in secondary operations. There is here little hope of improved speech. The closure of the roof of the mouth may be accom-

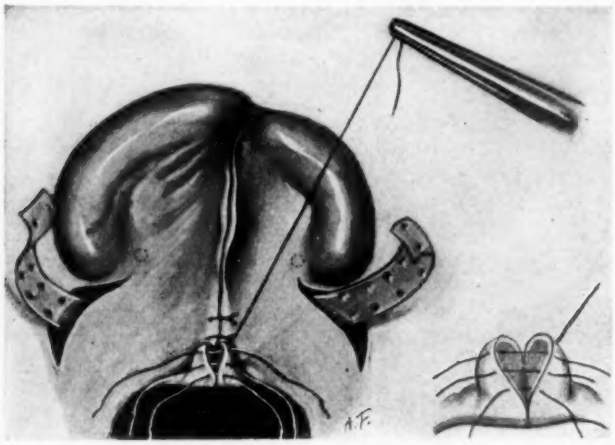

Fig. 14.—Stitching the posterior surface of the uvula and soft palate. Note that only the mucous membrane is included in the bite.

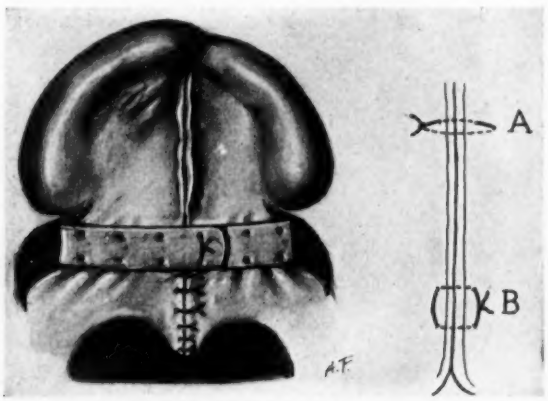

Fig. 15.—Tissue of hard palate united by mattress stitch. (*A*) Correct position of stitch; (*B*) incorrect position of stitch. It is obvious that *A* strangulates less tissue than *B*.

plished in many of these mutilated cases, and that at least relieves an annoying condition.

AFTER-TREATMENT

In the after-treatment two indications are paramount, namely, rest of the operative field as far as is possible and a full diet from the start. In infants no feeding is omitted. In older children abstinence

should not exceed six hours prior to operation, and orange juice and dextrose should be given two hours after operation. Symptoms of acidosis should be carefully watched for and, if found, counteracted at once. Absolute rest in this situation is impossible, but the principle should be kept in mind during the healing period. In order to establish better control, the child should be in the hospital for a few days prior

Fig. 16.—Operation completed. Tongue pressure obturator in position. Band crossed and tied with just enough pull to cause slight puckering of tissues of soft palate.

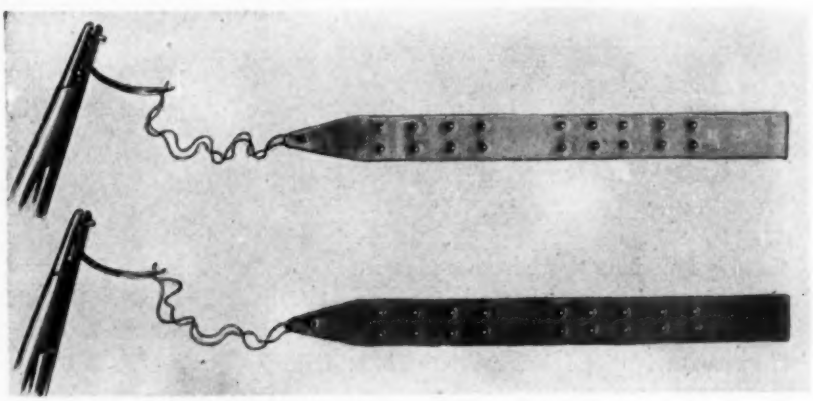

Fig. 17.—Lead and silver bands. Lead is preferable.

to operation and under a nurse trained in the care of children. This counteracts to some extent the defective training so prevalent in many homes. In no operation is control more important in order to secure the maximum of local surgical rest. Talking is prohibited in older children, and crying in younger ones should be reduced to a minimum.

The pernicious habit of tying the hands of younger children should be abandoned. If the child persists in putting his fingers in the mouth,

limitation of movement just short of obtaining that objective may be instituted. The question of the mother remaining with the child should be determined on its merits in each individual case. Under-feeding is a potent cause of unrest, especially in babies. Therefore, there should be no starvation with the object of preventing swallowing and mouth contamination. After feeding, a drink of sterilized water is given and the mouth cleansed with the same projected against the soft palate with a bulb syringe. A few drops of acriviolet solution (2 per cent) are dropped into each nostril and over the stitches on the palatal surface after each feeding. Sleep must be secured by opiates or sedative if needed. The more the child sleeps between feedings the better during the forty-eight hours following operation. The stitches,

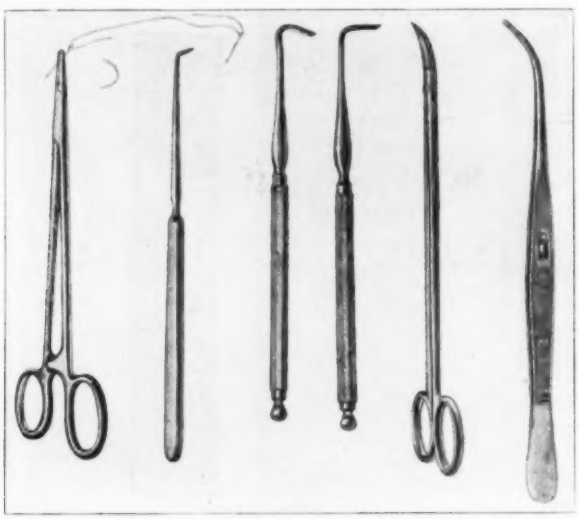

Fig. 18.—Instruments employed.

obturator and bands are removed on the eighth, ninth or tenth day, depending on the condition of the wound, and it is imperative that this is done under general anesthesia to avoid injury to loosely united tissues. A soft diet follows this period for about one week.

STATISTICS

In 1914, careful analysis was attempted of the patients operated on up to that time. Reports were fairly complete in sixty-four of ninety-two cases; only twenty of these patients were under 3 years of age. Seventeen of the thirty-eight patients under 6 years of age acquired good speech.

The cases in the second group have been less carefully analyzed due to inability to trace the patients. It is noteworthy that more than 95 per cent of the cases of cleft palate come (at least in my experience)

from the lower and often from the lowest strata of society. They might be called city nomads, so frequent and mysterious are their flittings. In this second group 80 per cent were under 3 years of age and most of these under 2 years, 15 per cent were between 3 and 6 years and the remainder were over 6 years.

TABLE 1.—*Summary of Data in Sixty-Four of Ninety-Two Cases Reported in 1914 and in One Hundred Six Cases Since 1914*

GROUP 1	
Under 3 years	20
3 to 6 years	18
6 to 10 years	9
Over 10 years	17
Complete failures	7
Partial failures	6
Deaths	1
Closed by first operation	37
Closed by second operation	12
Closed by third operation	1
Ultimate complete closures	50
Speech very good	17
Speech improved	17
Speech not improved	29
GROUP 2	
Under 3 years	82
3 to 6 years	19
Over 6 years	5
Complete failures	2
Partial failures	3
Closed by first operation	96
Closed by second operation	5
Ultimate closures	101
Deaths	0
Seriously ill	3
Of these, 61 were traced	
Speech much improved and in a few practically normal	36
Speech slightly improved	11
Speech poor or unimproved	14

TABLE 2.—*Cases Classified According to Extent of Lesion*

GROUP 1	
Soft palate only	12
Complete hard and soft palate	22
Soft palate and part of hard palate	18
Complete hard and soft palate with single harelip	4
Complete hard and soft palate with double harelip	2
Secondary cases; previous operations done elsewhere	6
	64
GROUP 2	
Soft palate only	23
Complete hard and soft palate	31
Soft palate and one half of hard palate	18
Soft palate and two thirds of hard palate	7
Single hard and soft palate with single harelip	18
Double hard and soft palate with double harelip	0
	106

In this group the percentage of closures shows a great advance over the first group due to earlier operation and to the use of the lead tension band. Of this number sixty-one were traced; about 60 per cent of these showed fair speech and all but one were derived from the class under 3 years of age and all in the class of primary closures.

Secondary operations destroy nearly all hope of improved speech. Not included in these lists are several secondary cases in which one or several operations had been done prior to consultation. I do not deem their inclusion a fair test of the technic here described. The good results are partly due to accepting mostly primary cases. Two patients with flat, wide clefts were not included, as on them a Lane operation was done. Excellent closures were obtained in most of the secondary cases, but no speech improvement.

I shall digress to mention an extraordinary condition, the parallel of which could not be found either in the literature or in the textbooks. A boy, aged 4 years, had extrusion of the premaxilla and double harelip with no visible cleft in the palate. The hard palate and alveolar arch had closed in behind the premaxilla, forming what appeared to be a perfect but diminished palatal arch. The deformity was almost irremediable. Enough of the premaxilla was removed, with all the teeth buds, to bring the lip into alignment, the clefts closed and the columella elongated.

Group 2 is divided into two subdivisions. The first shows the result immediately after operation. The second gives the speech results in the traced cases.

Comparing group 1 with group 2 a tremendous improvement is observed in the primary unions and a considerable advance in speech results. In group 1 the tongue pressure obturator only was employed. In group 2 both obturator and lead tension band were used. In group 1, only twenty children under 3 years of age were operated on. In group 2, eighty-two were under that age, and most of these were in their second year. I believe that the earlier the closure, the better the outlook for improved speech. The great majority of healed cleft palates are foreshortened. This is the chief cause of defective speech.

CONCLUSION

This operation should be undertaken only by those trained in the finer kind of plastic surgery. The technical details must be closely observed, and traumatism must be reduced to a minimum. The operation should be done prior to the eruption of the teeth, that is, at the end of the first or at the beginning of the second year. Speech training is of enormous help but difficult to secure in the social class from which these patients come. Cleft palate is a stigma of degeneracy and is often associated with a low degree of intelligence. This, too, is a factor in retarding speech. Until more is known about the prenatal influences which determine this stigma, one cannot hope to advance far beyond the present status in curing persons who have this humiliating defect.

TYPHOID NEURITIS OF THE RECURRENT NERVES WITH CONSEQUENT PARALYSIS OF THE LARYNGEAL DILATORS

REPORT OF A CASE *

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MONTREAL, CANADA

Cases of paralysis of the vocal cords as the result of infectious neuritis following typhoid fever are excessively rare. The majority of authors do not mention this complication, though they describe laryngo-typhus. Therefore, I believe it will be interesting to publish the following facts and to discuss the etiology of the lesions observed, recalling the causes capable of producing a paralytic disturbance of phonation.

REPORT OF A CASE

On Feb. 22, 1928, I was urgently called to the Notre-Dame Hospital to attend a patient who was choking. I learned that he had just been admitted in the service of Professor Lesage for typhoid fever dating from some time past.

On examination, I observed a double paralysis of the vocal cords, which were in the paramedian position. The glottis was reduced to a narrow cleft, and the dyspnea was alarming. The larynx was lightly infiltrated; however, there was no ulceration of the arytenoid cartilages, of the cavities and ventricular bands or of the epiglottis. There was no exolaryngeal lesion. The nasal fossae, pharynx and ears were normal.

In the presence of the state of suffocation, I immediately performed tracheotomy. The postoperative sequelae were normal, and the patient recovered his vitality.

From his mother and the physician who had treated him at first, I learned that there was no history of tuberculosis in his family. The young man was 23 years of age. At 3½ years of age he had had measles; at 5, scarlet fever; at 7, whooping-cough and at 13, pneumonia. At 8, his tonsils and adenoids had been removed. He had not, however, in the past suffered from laryngeal troubles.

As double paralysis of the posterior crico-arytenoid muscles is almost always due to a bulbar lesion of specific origin, I asked for a Wassermann test in spite of the absence of clinical symptoms. The report from the laboratory was that the reaction was negative.

Inquiring into the history of the typhoid infection, I learned that the onset had been about Jan. 10, 1928. The patient called on his physician on the 18th. His temperature then was 104 F. He was immediately sent back to his home,

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* Read before the Section of Oto-Rhino-Laryngology of the Canadian Medical Association, Montreal, June 20, 1929.

and was advised to remain in bed. He began to have delirium the following morning. He had probably been infected by his sister, who was living in the same house, and who had had typhoid fever one month prior to this. She had been contaminated by her sister-in-law, who had suffered from eberthian infection in the month of November, 1927.

The symptoms of my patient manifested themselves in severe form, and delirium was continuous. Although the respiration remained good till February 20, on this date it became more embarrassed. During the night of the 20th the respiration was bad, and the dyspnea increased. His physician decided to transfer him to the hospital on the 22nd.

Under the usual treatment for typhoid fever the patient improved and when he was well enough to get up, I asked for a roentgenoscopy. The examination revealed that there were no tracheobronchial ganglions, no mediastinal or esophageal lesions and no dilatation of the blood vessels.

The pulmonary secretions coming from the tracheal cannula were examined on two occasions, and no Koch bacillus was found.

One month after admission to the hospital, the patient had recovered from the typhoid fever, and the larynx, under the influence of rest, showed no more edema. However, the vocal cords remained in the paramedian position. It was only at about the eighth week that I observed a slight dilatation. From this time, the motions of abduction increased slowly. I prescribed strychnine and electrical treatment. As the improvement continued, on May 25, I began to obstruct the tracheal cannula in order to allow breathing through the larynx. The progress being favorable, the cannula was removed on June 18, and the wound was closed simply by coaptation, after the removal of some granular tissue. The stitches having been deeply placed, the healing was by first intention. The patient did not present any difficulty of respiration.

It is interesting to note that at the time of the departure of the patient from the hospital, on July 1, the dilatation of the vocal cords reached the intermediate position. The adduction of the two sides was normal. However, the voice was slightly hoarse. The patient, with a prescription of strychnine, went to the country, where he was to remain for a few weeks. His health was excellent. He was to report to me on his return.

COMMENT

I shall now summarize the principle causes that might have given rise to the paralysis of the larynx. I did not have to consider a cerebral lesion or psychic trouble.

Notwithstanding the age of my patient, and before making a complete clinical examination, helped by the laboratory, I had in mind the possibility of a bulbar involvement. However, the Wassermann reaction eliminated this hypothesis. There was no ground for considering the syndrome of the posterior jugular foramen or the more complex syndrome of the associated laryngeal paralyses. Indeed, the patient was neither syphilitic nor tuberculous. He never had had any trauma of the head or neck, and the mobility of the velum palatum, of the tongue, of the shoulder and of the sternocleidomastoid muscle was normal. The cervical region presented no abnormality capable of producing a compression of the recurrent nerve. No tracheobronchial adenopathy,

no mediastinal lesions and no dilatation of the blood vessels were to be observed. There was no tumor of the esophagus. So far as toxic neuritis was concerned, the work in the open air did not expose him to any intoxication; he used neither alcohol nor medicaments, and smoked only a few cigarets.

It remained, therefore, to consider only the infectious neuritis of the recurrent nerve. Certain infections occasionally produce paralysis of the larynx; the principle ones are diphtheria, typhoid fever, influenza, cholera, tuberculosis, syphilis, puerperal fever, acute articular rheumatism and even gonorrhea. With the exception of typhoid fever, my patient never had had any of these diseases. The disturbance of the laryngeal apparatus was manifested, as always in these cases, during the last period of the eberthian infection. After having been acute, as it was at first, and after having produced the paralysis of the dilators, the neuritis gradually disappeared. On treatment with strychnine and electricity, the vocal cords slowly recovered the power of dilatation as far as the intermediary position (constriction had never been impaired). It will be interesting to observe later whether or not the abduction regains the lateral position.

I do not have to insist that double paralysis of the dilators of the larynx represents a disease excessively severe, for the dyspnea that it provokes can rapidly end in death if tracheotomy is not performed.

It would be outside of my subject to discuss the value of Semon's law; numerous works have already been published in which authors have stated their opinions.

CONCLUSION

In the presence of the severe typhoid infection of my patient, the evolution of his disease, the state of improvement in which he is now, perhaps, his future cure, and the exclusion of the other conditions that might bring on a paralysis of the posterior crico-arytenoids, one cannot doubt that the laryngeal trouble was produced by a recurrent neuritis of eberthian origin.

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Clinical Notes

A CHART FOR REPEATED FUNCTIONAL HEARING TESTS*

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Innumerable history and examination forms have been used to record a single hearing test, and a few charts have been published with columns for four or five tests, but apparently in the past hearing tests have been used more for diagnosis than for recording the effect of treatment. From the time I first began to make use of hearing tests a need was felt for some means of recording not only the initial test but subsequent tests as well.

For several years a card was used with a column for each examination, dated across the top and a list of tests extending down the page on the left hand side. This was found to be a great help, but when treatment was prolonged over a long period, comparison of the various tests which had been made required laborious reviewing of the recorded numbers. This was found to be confusing. In several instances data of great importance recorded on these cards were not made apparent until the figures were charted as curves one above the other. To remedy this, the chart which is here presented has been arranged. Its use allows the effect of treatment, such as inflation and diathermy, to be seen at a glance. If each of the tests is carried out every time, they will be seen to tally with each other. Any lack of coordination will call for an explanation. It is difficult for a patient to fake each of the tests in the proper proportion. Many patients suffering from chronic progressive deafness fall into two psychologic types. The first have been told that nothing can be done and are fighting against accepting this verdict. These patients pass from physician to physician hoping for relief. They will deceive themselves and sometimes their aurist into believing that improvement is occurring after whatever treatment is used. After a variable period, both physician and patient finally decide that the improvement is more apparent than real. The other type of patient has been told by several physicians that he could be aided and has been disappointed until he has become a cynic and a "know-it-all." A carefully recorded hearing test plotted as a curve is essential to keep the physician's enthusiasm and the first class of patient's hopes from being misleading. A rapidly rising curve will convince the skeptic quicker than anything else.

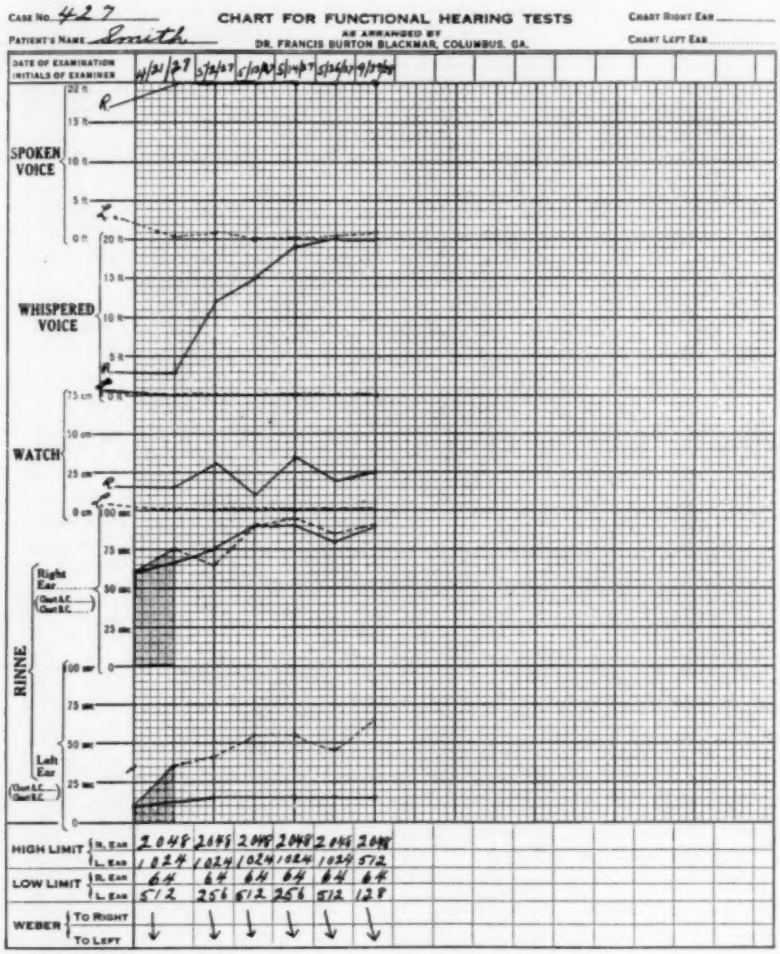
People are so constructed that they quickly forget the sufferings of the past. Thus, a patient may only be able to hear the spoken voice 3 or 4 feet away when treatment is begun and will say his hearing is only slightly improved even though treatment extends his spoken voice distance to 20 feet or more. In such a case, a graphic record of his past condition will be useful.

In the chart which I now use, space is provided for recording the distance at which the spoken voice can be heard up to 20 feet only, although normally the spoken voice should be heard at a distance of 40 feet. If the hearing for the spoken voice lies between 20 and 40 feet it will be demonstrated by a lowering of the distance at which a whisper can be heard. Few offices have provision for testing at a distance greater than 20 feet.

Passing down the column, below the spoken and whispered voice is the watch tick distance. Space is here allowed for a distance of 75 cm. or less. In case the particular watch used can be heard at 100 or 150 cm., it can be charted above the space for recording the watch tick and a line drawn inside the bracket for watch tick distance. It might not be amiss to say a few words about the

* Submitted for publication, June 1, 1929.

use of the watch as a test for hearing defects. Of the tests used in connection with this chart, the watch is probably the most reliable to indicate the presence of slight degrees of deafness, if it is used properly. In the first place, the same watch must always be used. The normal distance for the particular watch used must be known. I use centimeters in place of inches, because as this unit is not so familiar there is less likelihood of estimating "about so and so many"; in other



Sample of chart used for functional hearing tests, showing curves for patient with otosclerosis.

words, it almost forces one actually to measure the distance. A steel tape is drawn to the required distance. The windows usually must be closed to keep out foreign sounds. The zero end of the tape is held on the edge of the watch with the finger. The steel tape passes over the index finger placed behind the ear, and the distance is read off of the tape at this point. The case of the tape acts as a weight, drawing the tape tight as the watch is brought closer or further from the ear.

In the first three tests (spoken voice, whispered voice and watch tick), a dotted line is used for the left ear and a solid line for the right ear. For the initial test a line is drawn to the margin and labeled R. or L. to indicate the right or the left ear.

In charting the Rinne test, separate space is provided for the right and left ears. In this test, air conduction is shown by a solid line and bone conduction by a dotted line. The initial record of this test was found to be more graphic when the air conduction was charted on the first heavy perpendicular line and the bone conduction on the second heavy perpendicular line and these points connected. By filling in down to the zero line, a wedge is formed similar to that used for years by Dr. George Mackenzie of Philadelphia. The shape and height of this wedge indicate at a glance the type and degree of deafness as shown by the Rinne. If the slant of the wedge faces forward, there is nerve deafness; if the slant faces backward, there is conduction deafness.

Still further down the line, space is provided for recording the high and low tone limit for each ear.

The lateralization of the Weber test is indicated by an arrow with its head in the space for the right or the left ear, according to the side to which lateralization takes place. If the Weber test does not lateralize, a cross is made through both spaces.

The accompanying chart represents an actual case of otosclerosis in which the patient was treated for five months with iodine. The details of this treatment will be made the subject of a subsequent paper. The case is included here only to illustrate the use of the chart and not as a case history. The chart shows a steady and continuous rise in hearing for "the whispered voice" for the right ear, from 3 feet at the first examination to 20 feet at the last. The whispered voice could not be heard by the left ear. The spoken voice distance was 20 feet or better for the right ear and 6 inches for the left ear throughout. The watch tick varied from 15 to 35 cm. for the right ear. The watch tick could not be heard with the left ear. The Rinne test for the right ear showed an air conduction of sixty seconds and bone conduction of seventy-five seconds at the first examination. There was evidently some nerve deafness as well as conduction deafness, as shown by the reversed Rinne test and decreased bone conduction. There was some improvement in the nerve function, as shown by the rise of the bone (dotted line) and air conduction (solid line) to ninety seconds. In the left ear, bone conduction (dotted line) rose from thirty-five to sixty-five seconds, the air conduction (solid line) remaining at fifteen seconds. In the left ear, severe nerve deafness is the predominating condition. In advanced nerve deafness a reversed Rinne test is characteristic, as shown in this case, although the textbook picture of lowered bone and air conduction at their almost normal relation of 1:2 is correct for the lesser degrees of nerve deafness. The Weber test is recorded as lateralizing to the nerve-deaf ear throughout the period of observation. This indicates a conduction deafness which would be hidden by the severe nerve deafness, except for the evidence of the Weber test.

A PORTABLE NOSE, THROAT AND EAR TREATMENT CASE

ROBERT M. LUKENS, M.D., PHILADELPHIA

Many times the laryngologist is called on to attend patients who are confined to bed at home. These patients, under any circumstances, are difficult to treat properly because of the lack of facilities.

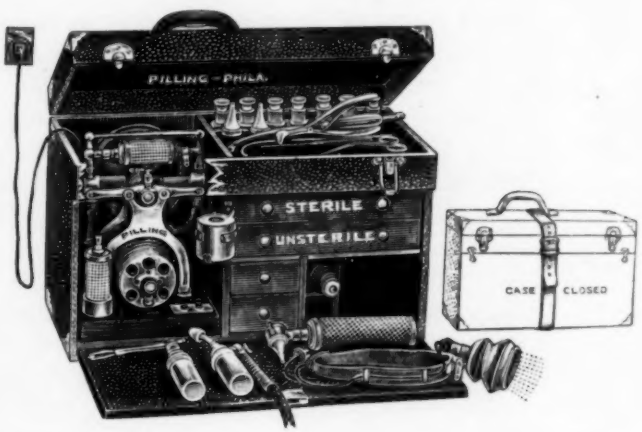
Cases in which there is a profuse purulent or mucopurulent discharge are the ones that give the most trouble. Treatment applied locally to the nose in such

cases has little effect. In the offices, these secretions can be readily removed by aspiration, and proper treatment can be applied.

The need for a suction apparatus in treating patients at home has been felt by most laryngologists, but because of the cumbersome apparatus required, the laryngologist usually has to content himself with swabbing instead of suction.

The portable treatment case illustrated has been designed to give the laryngologist a compact treatment case embodying the necessary instrument to take care of the majority of conditions of the nose, throat and ear requiring treatment at home. While it is probably larger and somewhat heavier than the average "specialist bag," I feel that the added advantages make it desirable.

The case measures approximately 18 by 8½ by 11½ inches (45 by 21 by 29 cm.) and weighs 20 pounds (9 Kg.) fully equipped. It is opened by raising the upper lid and dropping the front wall of the case, exposing to view a "treatment



Portable nose, throat and ear treatment case, opened to show equipment.

table" in the upper right part, containing bottles at the back. Beneath the treatment table there are copper drawers: The upper drawer is for instruments that have been sterilized, the lower drawer for the reception of instruments as they are used, later to be sterilized at the physician's convenience. Beneath these drawers there are two more drawers designed to contain special instruments, such as ear knives and tonsil knives. At the right of these drawers there are compartments to contain an electric otoscope, headlight or head mirror. The left side of the cabinet is occupied by Pilling's suction and pressure air pump; attached conveniently is a receptacle for sterile cotton. The pump is equipped with a cord and plug to fit in an ordinary outlet receptacle or other source of electric current.

This case is designed to be set up near the patient's bed, and to be opened and used exactly as the laryngologist would use his treatment table, pump, etc., at his office.

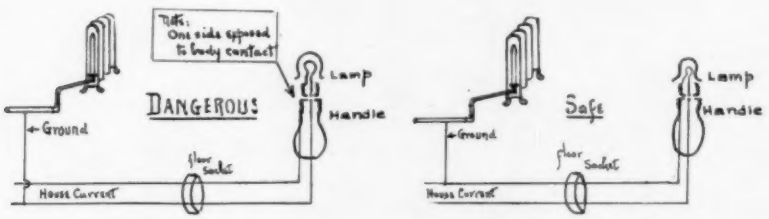
This case was made for me by Pilling & Son Co., Philadelphia.

1923 Spruce Street.

ELECTRICAL SHOCKS FROM DIAGNOSTIC INSTRUMENTS*

DOUGLAS MACFARLAN, M.D., PHILADELPHIA

The common practice, now prevalent, of illuminating diagnostic lamps by cutting down the "street" current, has led to the possibility of seriously shocking the patient during the examination. The house installations of the street current are invariably grounded "on one side" for the sake of fire protection. Further, there is usually a grounding of one side of the diagnostic lamp to the metal of the instrument, as it is much cheaper to construct these instruments in this manner. Now, if the attachment plug connecting the cabinet or wall plate with the house socket is so inserted that the grounded side does not coincide with the grounded side of the diagnostic lamp, the patient or the examiner will be shocked if any of the heating or plumbing fixtures in the office are touched. Frequently a phy-



Examples of dangerous and safe attachment of the diagnostic lamp.

sician or patient will turn on a cuspidor spigot, or will touch a radiator; they will then get a shock if they are in contact with the lamp at the same time.

I find that none of the medical supply houses know of this danger.

The possibilities of extravagant damage claims and lawsuits are obvious.

The same danger lies in turning on nearby electric lights while standing under the shower (or with women while curling the hair with an electric curling iron).

Proper correction of the circuits lies in the use of a plug and socket which will insure that the connections can be made in only one way, that is, with the ground of the house current coinciding with that of the lamp.

* Submitted for publication, Aug. 30, 1929.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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ADVANCES IN THE FIELD OF ALLERGY AS RELATED TO OTOLARYNGOLOGY DURING THE YEARS 1928 AND 1929*

W. W. DUKE, M.D.
KANSAS CITY, MO.

The following review of some of the literature on the subject of allergy for the years 1928 and 1929 is by no means complete and is reported chiefly for the purpose of showing the drift of thought in this line of work. The more interesting advances seem to me to have resulted from a study of sensitiveness to physical agents, especially from the studies reported by Thomas Lewis and his associates and by Horton and Brown.

I have gained the impression through reviewing the literature and through conversing with physicians who have interested themselves in this subject that the diagnosis and treatment of the allergic diseases is gradually molding itself into a well defined specialty. A great many of the larger medical schools have introduced a department of allergy.

The peculiar details of knowledge which seem essential to success in the study of cases of allergy and the materials and equipment which are required for accurate diagnosis and safe treatment are such that physicians who are not adequately equipped are likely to make errors which cause them to fear the subject. Several physicians of my acquaintance who have devoted a great deal of time to the study of allergy have discontinued work in this line because of reactions incident to pollen therapy. Reactions of this kind can be avoided or quickly controlled by physicians adequately experienced in the line but can actually menace the reputation of inexperienced physicians.

Recognition of allergic diseases seems to me to be essential to physicians, especially those who devote themselves to the practice of laryngology, dermatology and surgery and to the treatment of diseases of the chest but from the standpoint of recognition of the illnesses rather than from a standpoint of specific diagnosis and specific treatment.

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* Submitted for publication, Oct. 5, 1929.

Feinberg [1] reported an extensive and interesting review of the literature of 1927 and 1928 on the subject of hay-fever which can be highly recommended to the reader.

ETIOLOGY

Clarke, Donally and Coca,[2] continuing their studies of heredity in relationship to allergy, reported data which they believe show not only that the atopic tendency is inherited but also that the tissues to be involved are to a certain extent predetermined by heredity. In other words, patients who have asthma have more asthmatic antecedents than patients with simple hay-fever and vice versa, patients with hay-fever have a larger number of cases of hay-fever among their antecedents than patients with asthma do.

Bristow [3] stated that hay-fever and asthma are rather common in the negro race and quoted reports of a high incidence of the two conditions in South Africa and India. He stated the belief that variation in the statistics concerning the incidence of hay-fever in different districts is a matter of botanical rather than of racial difference.

Peshkin,[4] in a study of 285 children with asthma, ranging in age from 3 to 14 years, found a positive family history in 42.5 per cent. The time of onset he found to be more frequent at the age of 1 year than at other ages and stated that as children approach the age of 10, the incidence of onset becomes progressively less. The incidence of onset during the first five years is approximately the same for the group proved to be protein sensitive as for the group apparently not proved protein sensitive. He stated that patients with an onset of asthma after the age of 10 are relatively uncommon.

The incidence of positive family history is greater for children of 1 year than for any other age. The possibility of finding a positive family history after the age of 10 is progressively reduced. Peshkin stated that clinical and experimental data bring forcibly to light the fact that there exists a group of children in whom allergic diseases are acquired and in whom the lack of evidence on the influence of heredity is striking.

Inferences that can be drawn from this paper are interesting. Certain it is that in adults who apparently start having asthma after maturity

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1. Feinberg, Samuel M.: Progress in Hay Fever: A Review of the Hay Fever Literature of the Years 1927 and 1928, J. Lab. & Clin. Med. **14**:726, 1922.

2. Clarke, J. A. Jr.; Donally, H. H., and Coca, A. F.: Studies in Specific Hypersensitiveness: XXXII. On the Influence of Heredity in Atopy, J. Immunol. **15**:9, 1928.

3. Bristow, W. J.: The Ocular and Rhinological Manifestations of Allergy, J. South Carolina M. A. **24**:280, 1928.

4. Peshkin, M. Murray: Asthma in Children; Hypersensitiveness and Family History, Am. J. Dis. Child. **36**:89 (July) 1928.

a painstaking inquiry will often disclose a history of nasal, bronchial or skin symptoms in infancy. If Peshkin's observations are as broadly applicable as one would believe they should be, asthma is probably more frequently a congenital disease than would seem from the average history obtained in adults.

Storm Van Leeuwen [5] reported that in 10 per cent of the cases of asthma which he studied, sensitiveness to the acetylsalicylic acid group of drugs could be proved. He stated that this type of illness can be so severe that it is advisable in the treatment in cases of asthma to inquire for sensitiveness to drugs of the salicylate and antipyrine group and to the hypnotics before they are administered. He tried increasing tolerance for acetylsalicylic acid by giving small, gradually increasing doses. The results obtained were not favorable, he thought, and could prove actually harmful. He found, furthermore, that a large number of patients sensitive to acetylsalicylic acid were sensitive to other drugs as well, especially to hypnotics such as phenobarbital or to coal tar products, such as a brand of amidopyrine, U.S.P., and antipyrine. The symptoms that can follow the use of drugs in cases of this sort may be most severe and may last for many hours or several days.

Gibb [6] reported a case of anaphylaxis caused by a bee sting in which severe nasal and skin symptoms became immediately manifest and which were relieved by the timely use of epinephrine.

This case is reported because of the commonness with which severe shock symptoms follow the bites of insects in patients who happen to be specifically hypersensitive to them. This type of shock may be extremely severe and in two cases with which I am acquainted, death ensued within a few moments. As a rule, a huge hive around the site of the bite makes a diagnosis possible. Nasal, bronchial, skin and gastrointestinal symptoms frequently appear in all their glory and demand the immediate use of epinephrine in full dosage. Thereafter, the patient must be warned of the dangers attending exposure to the bites of certain insects.

Palmer and White,[7] from a study of 250 patients with cardiac asthma, stated that the grave prognostic significance of the condition is shown by the fact that in 170 of the patients, the average duration of life after the first attack of cardiac asthma was 1.4 years. The largest number of cases was found among the group with coronary disease,

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5. Van Leeuwen, Storm: Pathognomonic Significance of Hypersensitiveness to Asperin (Acetylsalicylic Acid), München. med. Wchnschr. **75**:1588, 1928.

6. Gibb, Douglas F.: Anaphylaxis from Pollen Introduced by Bee Sting, Canad. M. A. J. **19**:461, 1928.

7. Palmer, Robert S., and White, Paul D.: The Clinical Significance of Cardiac Asthma: Review of Two Hundred and Fifty Cases, J. A. M. A. **92**:431 (Feb. 9) 1929.

hypertension, or both (10.7 per cent), specific heart disease (21 per cent) and chronic nephritis (19 per cent). The actual mechanism for the production of asthma in cardiac cases did not appear clear, but left ventricular failure was an apparent manifestation. The coincidence of poor heart sounds, gallop rhythm and alternating pulse, as a rule, gives a bad prognosis. Aortic regurgitation was the only common valve defect. Rest and the administration of digitalis apparently were effective in reducing the number and severity of attacks. In treatment, nitrites, alcohol and morphine were also recommended.

Bishop and Kendall [8] studied the effect of electrical stimulation on smooth muscle of the intestine of animals when the outside of strips of intestine was washed with solutions containing histamine or solution of formaldehyde or containing homologous protein in sensitized animals. The concentration of the wash solutions was below the threshhold for stimulating contractions in themselves. These authors found that histamine solutions and homologous protein solutions increased the responses to electrical stimulation whereas solution of formaldehyde decreased them.

These observations are important in relation to so-called secondary factors in the pathogenesis of allergic phenomena. If an analogy can be assumed to exist between cases in animals and those in human beings, one would surmise from the foregoing results that nonspecific irritants can add to the severity of allergic phenomena. This does not indicate, however, that physical agents per se are not potential factors in eliciting similar phenomena.

POLLEN STUDIES AND TEST METHODS

Lamson, Piness and Miller [9] found that skin sensitiveness is reduced by treatment with pollen extract and that this persists for weeks or months or even years. They also made the observation which harmonizes with clinical experience generally that treatment with extracts to which the patient does not react does not sensitize them. Human beings differ in this respect from animals such as the guinea-pig, which can be sensitized with great ease with almost infinitesimal doses of foreign protein. These observations, confirmed by clinical experience in general, agree with opinions expressed many years ago that it is

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8. Bishop, G. H., and Kendall, A. I.: Effects of Histamine, Formaldehyde and Anaphylaxis upon Responses to Electrical Stimulation of Guinea Pig Intestinal Muscle; Agents Applied to Serous Aspect of Intestine, Am. J. Physiol. **85:** 546, 1928.

9. Lamson, R. W.; Piness, G., and Miller, H.: Pollen Allergy; Skin Reactions of Patient Before and After Treatment with Spring (Grass) Pollens, Am. J. M. Sc. **175:**791, 1928; Pollen Allergy with Special Reference to Skin Reactions Before and After Treatment with Fall Pollens, ibid. **175:**799, 1928.

difficult or impossible to sensitize a human being. The importance of this idea is by no means small. If it were possible to sensitize a human being, one would scarcely be justified in testing sensitive persons with large numbers of foreign substances or in treating patients with vaccines or other extracts containing protein. If one rarely or never sensitizes a human being by skin testing, and this seems the case, one is justified in continuing its use.

Piness and McMinn,[10] and also Wodehouse,[11] discussed at length the structure of the different types of pollen and suggested methods for their identification. It would be impossible in an article of this scope to do justice to these careful studies.

Kahn [12] stated that skin tests to pollen, even when made intradermally, are frequently negative in young children suffering from pollen asthma. The commonness of food asthma in infants, the relative uncommonness of pollen asthma in infants and the rarity of pollen asthma in young people in the absence of nasal symptoms make one doubt the correctness of Kahn's diagnosis of pollen asthma in children. Cases of true pollen asthma give positive pollen tests in such a gross majority of adults that one is always inclined to doubt the correctness of a diagnosis of pollen disease in the absence of a positive skin test. The behavior of the skin in the case of pollen sensitiveness is grossly different from the behavior of the skin in food sensitiveness in this respect. In the latter type of case, tests with foods are more commonly negative than positive even in proved cases. Kahn's experience with pollen tests in cases proved to be pollen sensitive is grossly different from mine.

Huber,[13] in testing patients with specimens of the same pollen gathered in different geographic locations, stated that some patients consistently give greater reactions to the specimens collected in one locality than to the specimens of other districts. Physicians have frequently wondered whether or not patients can be tested and treated best by

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10. Piness, G., and McMinn, H. E.: The Rôle of the Structural Features of Pollen Grains in Identifying the Most Important Hay-Fever Plants of California, J. Lab. & Clin. Med. **12**:1164, 1927.

11. Wodehouse, Roger P.: Pollen Grains in the Identification and Classification of Plants: III. The Nassauvinae, Bull. Torrey Botanical Club **56**:123, 1929; Pollen Grains in the Identification and Classification of Plants: IV. The Mustisieae, Am. J. Botany **16**:297, 1929; Pollen Grains in the Identification and Classification of Plants: L. The Ambrosiaceae, Bull. Torrey Botanical Club **55**:181, 1928.

12. Kahn, I. S.: Significance of Negative Skin Tests in Pollen Hayfever and Asthma in Infants and Young Children; Treatment, South. M. J. **21**:559, 1928.

13. Huber, Harry L.: The Variability of Skin Reactions to Pollens, paper read before the American Association for the Study of Allergy, Minneapolis, Minn., June 12, 1928.

products collected in districts in which they have clinical symptoms. It is certain that good diagnostic and therapeutic results can be obtained in a large number of patients through the use of pollen collected in areas that are different botanically from the one in which they reside. Whether or not they would do better with specimens collected in their own locality remains an open question. Huber's results would suggest that the source of pollen is a matter of minor importance.

Brown [14] reported a rather surprising result in finding not only that skin tests to giant and short ragweed are approximately identical but that in patients treated with ascending doses of one extract, the other can be substituted without the production of a constitutional reaction. This, in a way, harmonizes with Kahn's results reported several years ago in which patients sensitive to Bermuda grass were actually desensitized by an extract of Sudan grass. The two plants are similar only in the sense that both are grasses. The difference in appearance and size of these two grasses is far greater than the difference between large and small ragweed.

Chobot,[15] in comparing the atopens of timothy pollen with Bermuda grass, found that each of ten patients sensitive to timothy who were never apparently exposed to Bermuda grass gave equal reactions both by skin tests and by conjunctival tests to extracts of the two pollens. They found further that passive desensitization of locally sensitized areas could be accomplished through the use of either timothy pollen extract or Bermuda grass extract when the transfer was made from patients suffering from timothy hay-fever. He advised patients with timothy hay-fever who expect to spend the winter months in the south to continue the treatment with timothy extract in order to prevent reaction against Bermuda grass pollen.

From a comparative study of skin and ophthalmic tests for sensitiveness to horse serum, Spicer [16] concluded that a positive ophthalmic test obtained with undiluted horse serum, while not so sensitive as an intradermal test with diluted horse serum, is a more dependable guide for clinical sensitiveness. Many patients who tolerate horse serum give a positive skin test. In fifty-nine patients who reacted to horse serum, each of the six serious reactions resulted in patients who gave a positive

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14. Brown, A.: Studies in Hypersensitiveness; on the Question of the Identity of the Atopens of the Pollens of High Ragweed and Low Ragweed, J. Immunol. **13**:73, 1927.

15. Chobot, Robert: Studies in Specific Hypersensitiveness: XXVIII. The Relationship of the Atopens in the Pollen of Bermuda Grass (Cynodon Dactylon) and Timothy Grass (Phleum Pratense), J. Immunol. **16**:281, 1929.

16. Spicer, Sophie: Ophthalmic Test for Horse Serum Sensitivity Contrasted with the Intradermal Performed on Three Hundred Fifty-Three Patients, J. Immunol. **15**:335, 1928.

ophthalmic test. Many of the patients who gave positive intradermal tests had mild serum reactions. Spicer recommended the slow administration of antitoxin, if indicated, in small divided doses in patients suspected of serum sensitiveness.

AUTOPSY REPORTS

Kountz and Alexander [17] added three to the meager number of autopsy reports of patients who died of asthma. They confirmed Huber and Koesslers' observation of thickening of the peribronchial muscle and of the subepithelial layers of the bronchial membranes and Fashingbauer's observation of dilatation of the small bronchi. They also found rupture of the basement membrane, infiltration and destruction of muscle by eosinophils and destruction of bronchial cartilage. A similarity between the lesions in the nasal mucous membrane and those of the epithelial and subepithelial layers of the bronchi was also discovered.

EFFECTS OF ALLERGY

Rowe [18] reported further on food allergy as a cause of abdominal pain, gastro-intestinal manifestations, urticaria, angioneurotic edema, eczema, migrane, asthma, epileptiform attacks, perennial hay-fever and nasal congestion, bladder pain, hypotension and unexplained fever. He stated that food allergy must always be considered as a possible cause of manifestations of the foregoing types in the absence of other adequate cause for the disorder, especially in patients who give a family or personal history of allergic symptoms or a history of dislike or disagreement of certain foods. The diagnosis can usually be made through the use of skin tests or through the use of diets such as he has arranged and called "elimination diets." Rowe found the latter especially useful since in food allergy skin tests are frequently negative.

Beecher [19] reported, in agreement with previous reports by Rowe, that migraine is frequently a result of allergy. He stated that cases of migraine frequently present a family history of allergy, that protein tests are frequently positive and that in such cases, withdrawal of certain foods is likely to give complete relief. He stated that the results in general are so good that physicians should refrain from giving a bad prognosis in the case of family migraine.

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17. Kountz, W. B., and Alexander, H. L.: Death from Bronchial Asthma; Report of Three Cases, Arch. Path. **5**:1003 (June) 1928.

18. Rowe, Albert H.: Food Allergy; Its Manifestations, Diagnosis and Treatment, J. A. M. A. **91**:1623 (Nov. 24) 1928.

19. Beecher, W. L.: Migraine or Sick Headache: A Sensory Disturbance Due to Protein Sensitization or Idiosyncrasy, Illinois M. J. **55**:123, 1929.

Hollander [20] urged that attention be paid to the importance of allergy by physicians prescribing diets to patients who give a personal or family history of allergic disease.

Robinson [21] reported a case of uterine spasm caused on three occasions by treatment with orris root. This observation is interesting in relationship to treatment of pregnant women with foreign agents, such as pollen.

Vallery-Radot,[22] in an interesting article, stated that what is spoken of as allergic coryza and bronchial asthma are two syndromes that are of an identical nature, may have an identical etiology, and demand identical treatment. These two syndromes, he said, may be associated together, one may alternate with the other, or one may replace the other. The two types of illness differ only in the fact that they have different points of localization.

Vaughan [23] reported a series of eleven cases of mucous colitis which he believed he proved to be allergic in origin. Seven of the patients were benefited symptomatically through specific protein avoidance. He stated that treatment did not cause spectacular results, but he attributed this to the fact that secretory alterations had taken place which interfered with the immediate recovery on removal of the specific cause of the ailment.

Bivings [24] reported a case of death in an infant, aged 10 months, apparently caused by uncomplicated asthma. This case is reported because of the rare occurrence of death caused by uncomplicated asthma, especially in children. The complications are more prone to terminate fatally.

Hazen [25] discussed allergic dermatoses, and under this title he included the following list of diseases: allergic dermatitis (which may be either local or general), urticaria, erythema multiforme, purpura, neurodermite and simple lichenification, some cases of rosacea, dermatitis herpetiformis and infantile eczema.

He mentioned as causes cosmetics, drugs, pollens, dander, dust, wearing apparel, pillows, mattresses, insecticides, dyes, foods, colonic absorp-

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20. Hollander, Edward: The Importance of Allergy in Prescribing Diets, Am. Med. **23**:918, 1928.

21. Robinson, E. M.: Report of a Case of Uterine Spasm Caused by Orris Root, M. J. & Rec. **129**:139 (Feb. 6) 1929.

22. Vallery-Radot, L. Pasteur; Blamoutier, Pierre, and Justin-Besançon, L.: Spasmodic Coryza, Equivalent of Asthma, Presse méd. **36**:625, 1928.

23. Vaughan, Warren T.: Allergic Factor in Mucous Colitis, South. M. J. **21**:894, 1928.

24. Bivings, Lee: Fatal Bronchial Asthma in a Ten Months Infant, Arch. Pediat. **45**:369, 1928.

25. Hazen, H. H.: Allergic Dermatoses, Arch. Dermat. & Syph. **18**:121 (July) 1928.

tion, vocational exposure, bacterial substances, irritability of the vegetative nervous system, physical agents such as light and cold, and endocrine disturbances.

He stated that cutaneous tests do not have the same value in the diagnosis of allergic dermatoses that they have in hay-fever and asthma. His experience and that of Bernton, whom he quoted, harmonizes in this respect with my own. In my experience, a few of the negative tests obtained in dermatologic cases are due to the fact that frequently the manifestation, eczema, is a delayed reaction. Consequently, the result of tests usually gives a delayed reaction. Furthermore, tests can be applied better through the direct application of an untreated suspected agent than through the injection of solutions. Some failures in diagnosis are due to failure to test with physical agents. Testing with physical agents, such as light, heat, cold and mechanical irritation should be varied according to the patient's history. An effort should be made to imitate the type of activity or the type of exposure which, by history, makes the patient ill. If this is done, a large number of cases will give positive results on physical tests and a diagnosis as to the cause of the disorder will be possible. One should not hesitate to say that heat, cold or light is a cause of an illness if he can consistently reproduce the illness by the appropriate application of heat, cold or light.

Criep and McElroy [26] reported finding normal blood calcium in atopic hypersensitiveness. They also stated that calcium therapy does not give rise to a permanent increase in blood calcium in atopically sensitive patients. They found that the metabolic rate was not reduced. They noted, however, the frequent presence of achylia or subacidity.

SPECIFIC TREATMENT

In studying the pollen content of still air indoors as compared with the outside air and outside air behind the propeller of an aeroplane, I [27] found that the amount of pollen deposited on a unit of surface in a room kept constantly closed for one year was on an average ½ grain (0.03 Gm.) per day as compared with 56 grains (3.6 Gm.) per unit in a room that was well ventilated. In the ventilated room, the counts were relatively high because of the fact that pollen extracts were occasionally made in this room.

In comparative counts made under a different set of conditions on a given day, the pollen deposited on a unit of surface in a room closed for

26. Criep, Lee H., and McElroy, W. S.: Atopy: Blood Calcium and Gastric Analysis, Arch. Int. Med. **42**:865 (Dec.) 1928.

27. Duke, W. W.: The Pollen Content of Still Air, in Relationship to the Symptoms and Treatment of Hay-Fever and Asthma, J. A. M. A. **91**:1709 (Dec. 1) 1928.

many days was zero. In a room closed one day only, the count was 3. In a room that had been closed for one day only but in which a fan had been used for twelve hours, the count was 8. In a room that was ventilated, the count was 20. On the same day, the count in the outside air was 191. In contrast with this, the amount deposited in the air behind the propeller of an aeroplane revolving at full speed averaged more than 300 in a period of thirty seconds as compared with an average of 0.6 deposited simultaneously and during the same period of time on a slide kept in the open air on the ground. The amount of pollen deposited up to a height of 3,000 feet was approximately the same as that deposited on the ground. Above this point, the air was apparently free of pollen.

These results seemed important in relationship to the treatment of pollen disease, for it seems evident that one would inhale more pollen outdoors than indoors and that during the pollen season sensitive persons would do best indoors and should avoid the undue exposure incident to trips on a train or in an automobile. Furthermore, it tends to account for recurrences and failures in patients receiving careful treatment. Those whose habits or occupation keep them indoors during the pollen season are less intensely exposed than those whose occupations keep them in the open air or on moving objects. The factor, exposure, has to be taken strictly into account in treated patients for, as a rule, treatment by the best of physicians does not increase tolerance for unlimited exposure to pollen.

A number of papers appear yearly on the pollen treatment of pollen hay-fever and asthma. Although successful methods used in different localities vary, the beneficial results obtained by different authors seem remarkably uniform. One rarely sees an unfavorable report by investigators who are adequately experienced to give successful treatment in the cases observed in their own districts. I [28] can say from personal experience that methods used successfully in other districts do not give results that are satisfying to the average patient in this district. This is apparently due to the fact that the pollen concentration of the air in the inland cities is unbelievably gross as compared with the pollen concentration in the air of towns on the seacoast such as New York, Richmond, San Francisco or Los Angeles. In fact, a person residing in an inland city, such as Kansas City, wonders why pollen hay-fever should exist in cities on the coast. A majority of patients living in a district such as this can obtain relief without treatment on visiting districts where the pollen concentration of the air is as small as it is in New York or in Richmond, Va.

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28. Duke, W. W.: Asthma, Hayfever, Urticaria, and Allied Manifestations of Allergy, St. Louis, C. V. Mosby Company.

Almost all observers agree that the results of pollen therapy give relief for the season in patients who are adequately treated but that the results are not often permanent. The recent papers of Chamberlain, Piness, Bernton, Vander Veer, Cooke and Spain, Conway, Foss, Freeman, von Bansky, Strebel and Schwartz are interesting in this respect.

In accounting for causes of failure, Feinberg [1] mentioned: the inexperience of physicians with this form of therapy; the failure of early preseasonal treatment carried into the season; the failure of individualization of dosage of pollen and individualization of time interval according to a patient's reaction; the failure of careful attention to the species of pollen which is used and finally, lack of care in preservation of the potency of the pollen extract that is used. Cohen, Reicher and Breitbart [29] attributed the cause of failure to inexperience and improper treatment, to the use of nonpotent extracts, inadequacy of final dosage and, finally what seems to me to be important, to contact with unusually large doses of pollen.

The cause last mentioned by Cohen, Reicher and Breitbart seems worthy of emphasis. Patients in whom treatment has given a degree of tolerance that is adequate to protect them against doses of pollen such as are encountered indoors or on rainy, cloudy, still days may not be protected against doses encountered during a trip in an automobile or on a train or may not be protected against the quantity encountered on windy sunny days at the height of the season. One is most surely destined to disappointment if he does not carefully warn his patients under treatment against undue exposure to pollen.

Ramirez,[30] in reviewing the effect of different methods of treatment in 425 cases, stated in substance that he believes that it is important to give a large amount of pollen before the onset of the pollen season. He stated that it is quite evident that it is essential to continue treatment throughout the season. He stated further that local desensitization increases relief and that autogenous vaccines given together with pollen are helpful. He thought that the Kromayer lamp helps some patients and has no effect on others and that, in general, it is more useful in nonspecific vasomotor cases than in specific pollen cases. He stated further that vaccines used alone in cases of pollen sensitiveness are of no value and that local pollen treatments are useless unless given in connection with pollen injections. He saw no particular advantage in the use of a large number of pollens in therapy except for the fact that, as

29. Cohen, M. B.; Reicher, J., and Breitbart, J.: Causes of Failure in Hay Fever Treatment, Ohio State M. J. **24**:372, 1928.

30. Ramirez, Maximilian A.: Statistical Report on the Treatment of Hay Fever, Am. J. M. Sc. **176**:856, 1928.

a rule, in this type of therapy larger doses of pollen protein are injected. He believed that the optimum method of therapy consists in preseasonal treatment extending over a period of about three months with two weeks' intensive treatment just prior to the season, as high a dose as possible being reached, with the continuance of pollen injections at two week intervals throughout the season and finally, with local treatment with pollen solutions in the nose and eyes in increasing concentration both preseasonally and seasonally. Mild symptoms, such as may appear in patients treated in this manner, can be easily controlled through the use of ephedrine, especially if it is combined with small doses of codeine or atropine.

Rackemann [31] reported a summary of 410 cases of hay-fever in which relief was obtained by pollen treatment, stating that, whereas seasonal relief was obtained, the result was not permanent in more than 6.3 per cent of the cases. In these the duration of relief had lasted for an average of 4.6 years. Permanent relief apparently did not depend on the number of years of treatment, on the particular technic used in treatment or on the disappearance of the skin reaction. Rackemann believed that cure is a clinical rather than an immunologic phenomena.

Black [32] reported further on the oral administration of pollen extract. In patients treated by doses gradually increasing from 10 drops of a 5 per cent extract by mouth three times a day up to sixty drops three times a day the results were good but not so good as those obtained by hypodermic treatment.

NONSPECIFIC AND SYMPTOMATIC TREATMENT

Ling [33] reported that his results in the treatment of children having asthma with tuberculin are not striking and do not harmonize with those of Van Leeuwen, who claimed that in 50 per cent of the cases relief can be obtained by this means. He thought it justifiable to state, however, that the frequency and the intensity of asthmatic attacks can be diminished by tuberculin therapy.

Ling was careful in his work to exclude pulmonary fibrosis and enlarged mediastinal glands from the cases reported.

Vallery-Radot and Giroud [34] employed peptone in the treatment of hay-fever and reported good results. They used 0.1 cc. doses of a 50

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31. Rackemann, Francis M.: The Ultimate Prognosis in Hayfever, Am. J. M. Sc. **177**:333, 1929.

32. Black, J. H.: Oral Administration of Pollen; Clinical Report, J. Lab. & Clin. Med. **13**:709, 1928; The Oral Administration of Pollen, ibid. **12**:1156, 1927.

33. Ling, T. M.: Treatment of Asthma in Children with Tuberculin, Lancet **2**:972, 1928.

34. Vallery-Radot, P., and Giroud, P.: Survey of Recent Studies in Hay Fever, Paris méd. **1**:151, 1928.

per cent solution of Witte's peptone and increased this according to indications. This method of treatment is ineffective in this district. I can find no other grounds for disagreement with these observers.

Ramirez [35] stated that after a careful and unbiased analysis of patients treated with peptone he felt justified in stating that, in his experience, peptone is of no value in the treatment of bronchial asthma. This statement seems rather strong.

Rackemann and Scully [36] reported good results from the use of vaccines in the treatment of asthma and so-called colds. From a study of 346 patients with asthma treated with vaccines, the writers obtained a good result in 74 per cent. In the prevention of colds, they obtained a good result in 68 per cent of 101 patients. They stated that the results are temporary but can be repeated. They stated further that stock vaccines seem to be as effective as autogenous vaccines. They stated that occasionally the same patient is benefited by different types of vaccine but that occasionally they are benefited by one and not by other types of vaccines. For this reason, they are inclined to believe that in many instances the action is nonspecific while in others there is a certain degree of specificity in the action of the vaccine. They were unable to show that one organism was more important than another.

Vallery-Radot [37] reported relief from attacks of spasmodic asthma in suitable cases by venous section. He did not attempt to account for the relief but simply stated that in a certain number of cases bleeding may stop the attack.

Malone [38] believed that relief which is obtained in a certain proportion of so-called nonsensitive types of bronchial asthma in high altitude is due probably to the low moisture content of the air.

A number of papers appear yearly on the use of ephedrine in the treatment for allergic conditions, and practically all the authors agree that whereas it has a sphere of usefulness, this is seriously impaired, if the drug is given in full dosage, by such symptoms as weakness, tremor, nervousness and sleeplessness. A 3 per cent solution used as a nasal spray and eyewash gives almost universally good results. Gay and

35. Ramirez, Maximilian A.: Peptone Treatment in Bronchial Asthma, Arch. Int. Med. **42**:368 (Sept.) 1928.

36. Rackemann, Francis M., and Scully, Margaret S.: Studies in Asthma: III. The Use of Vaccines in Asthma and "Colds," New England J. Med. **199**: 314, 1928.

37. Vallery-Radot, P.; Blamoutier, P., and Rouquès, L.: Relief of Certain Asthmatic Crises by Venous Section, Bull. et mém. Soc. méd. d. hôp. de Paris **52**:1191 (July 19) 1928.

38. Malone, J. T.: Relationship of Altitude to Bronchial Asthma, U. S. Vet. Bur. M. Bull. **4**:1032, 1928.

Herman,[39] in a report of 100 cases of asthma in which ephedrine was used, stated that the drug given by mouth produces its most satisfactory result when administered at the onset of a paroxysm and that relief can be obtained most quickly in patients whose symptoms are due to a specific foreign protein. Eight patients failed to derive benefit from the drug. Those authors concluded that ephedrine adds greatly to the relief of persons suffering with asthma but that it cannot replace epinephrine in the treatment of an acute paroxysm.

The reports of a large percentage of cases of pollen hay-fever and asthma in which complete relief was obtained by the use of agents such as calcium by Pottenger, phosphoric acid by Sterling [40] and of acids by Beckman [41] seem surprising. I try to account for this on the basis of the fact that all these observers live in districts where the pollen concentration of the air is low as compared with that of the Middle West. Beckman, after a study of treatment of hay-fever by acids, went so far as to say "the suggestion is therefore offered that a clean start be made in the study of allergy, the new researches to be based upon the assumption that the allergic individual is one who is at all times in a state of relative alkalosis." I have observed benefit from the use of very dilute acid solutions used locally in the nose and conjunctival membrane and from the inhalation of acid gases, but I have rarely obtained a pronounced result from the internal administrations of hydrochloric acid, phosphoric acid, or hydrochloric acid yielding compounds.

SENSITIVENESS TO PHYSICAL AGENTS

Thomas Lewis [42] recently published a monograph in which he reported exhaustive studies on the responses of the skin vessels to stimulants and irritants of many varieties. These studies are interesting in relation to the respiratory mucous membrane because the laws which govern the action of the capillaries of the skin probably also govern surface vessels in general and therefore probably apply to the membranes of the nose and bronchial tract.

Under the influence of an injury such as can be applied by stroking the skin with a rough instrument, certain small vessel responses can be

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39. Gay, Leslie N., and Herman, Nathan B.: Treatment of One Hundred Cases of Asthma with Ephedrine, Bull. Johns Hopkins Hosp. **43**:185, 1928.

40. Sterling, A.: Value of Phosphorus and Calcium in Asthma, Hay Fever, and Allied Diseases, J. Lab. & Clin. Med. **13**:997, 1928.

41. Beckman, Harry: The Acid Treatment of Hay Fever, M. J. & Rec. **130**:9 (July 3) 1929; Nitrohydrochloric Acid in Hay Fever, Wisconsin M. J. **27**: 253, 1928; Nitrohydrochloric Acid in Treatment of Hay Fever, Am. J. M. Sc. **174**:525, 1927.

42. Lewis, Thomas: .The Blood Vessels of the Human Skin and Their Responses, London, Shaw & Sons, 1927, pp. 322.

elicited in normal persons which have been designated by previous writers as dermographia. In certain persons, these responses are much exaggerated and give rise not only to redness of the skin but to a localized type of edema which has been termed by previous writers as factitious urticaria. In describing the vessel reactions in this type of urticaria, Lewis used the term, triple response. The first phase of the triple response consists of reddening of the skin along the line of the stroke. This is caused by local capillary dilatation. It is quickly followed by reddening of the skin over a diffuse area around the stroke. This second phase of the reaction Lewis called a flare. Finally, as a third phase of the triple response, edema appears and is confined to the area of the first reddening—that is, to the area directly irritated by the stroke. I [43] had previously called this type of urticaria urticaria dermographia to distinguish it from an exactly similar type of reaction caused in other patients by light (called urticaria solaris), and in another type caused specifically and only by cold (called urticaria hiemalis). All three reactions, as a type, were called contact reactions because of the fact that edema was confined to the point of contact between the skin and the specific physical agent that caused it. The term was used to distinguish it from other types of urticaria caused by physical agents in which the reaction occurred both at the site of contact and in distant structures as well or in some cases, at distant structures only and frequently at some point in the respiratory mucous membrane. The entire group of cases were classified under the term physical allergy, in order to separate them from similar reactions caused by sensitiveness to material agents, such as pollen, egg or milk.

According to Lewis' view, the first and third phases of his triple response are caused by the action of histamine or histamine-like substances (which he calls H substance) on the small vessels themselves, causing dilatation and giving rise to redness and increased permeability of the vessel walls. The latter causes local edema. Lewis stated the belief that H substance is liberated by disintegration of protein molecules caused by the stroke injury. Similar reactions of similar origin and caused by H body, he believed, can be caused also by thermic injury or chemical injury or by the effect of allergen in persons sensitive to allergen. Through the latter assumption, he connected this interesting type of urticaria caused by physical agents with the urticaria caused by sensitiveness to the material substances, such as egg or milk. H substance, he assumed, can act directly on small vessels causing dilatation and edema and through local nerve mechanisms can cause diffuse dilata-

43. Duke, W. W.: Urticaria Caused Specifically by the Action of Physical Agents (Light, Cold, Heat, Irritation, and Physical and Mental Exertion), J. A. M. A. **83**:3 (July 5) 1924.

tion or flare. With the liberation of large amounts of H body, constitutional reactions manifest themselves. This is observed when large areas of skin are irritated in highly sensitive persons. This view is not at odds with the favorite of four possible hypotheses which I suggested in an endeavor to account for the fact that a physical agent, such as light in one type of patient, cold in another or scratches in another may give identical manifestations which resemble minutely the reactions caused by pollen in persons sensitive to pollen or by egg in those sensitive to egg. My theory of physical sensitiveness seemed in harmony with Coca's theory for pollen sensitiveness. Coca assumed that a pollen reaction is caused by contact between atopen and its related atopic reagin in a person of atopic heredity. My theory for physical sensitiveness was based on an assumption that a new chemical body can be formed in certain persons under the influence of a physical agent such as light, heat or cold and that if the person happened to be of atopic heredity, he might become specifically hypersensitive to this new chemical body and thereafter react to it in exactly the same way that a person of atopic heredity may become hypersensitive to pollen and thereafter react to it on appropriate contact. According to this view, therefore, patients specifically sensitive to certain physical agents react in exactly the same way that a person sensitive to pollen reacts to pollen, the chief difference being that the pollen sensitive person reacts to a ready made substance whereas the person sensitive to a physical agent reacts to substances as they are formed in his own tissues by the physical agent to which he reacts. According to Lewis' view, one would have to assume that the reaction between atopen and atopic reagin results in a destructive effect on proteins with the liberation of H body and, similarly, in accounting for physical sensitiveness, one would have to assume that the reaction of the patient's tissues to the new body formed by the physical agent would do so through the breaking down of protein with the liberation of H body.

Many physicians have, for a number of years, been looking for a relationship between histamine-like bodies and allergic or atopic reactions. Lewis' monograph is based on extensive clinical research and if points can be proved by indirect evidence, it must be granted that Lewis has done a great deal toward demonstrating this point. The similarity between atopic reactions and histamine reactions is so exact in so many respects that it is hardly possible to disregard an idea that there is a direct relationship between the two, especially since it is a known fact that histamine-like bodies can be liberated by a destructive or normal catabolic action on normal proteins. Lewis' view, if true, might easily account for the fact that similar manifestations can follow the effect of such varied agents as light, heat, cold and friction in certain sensitive

persons and that such manifestations might be identical in origin with those produced by pollen in persons sensitive to pollen or egg in those sensitive to egg. Further study will no doubt simplify the present theories and will enable one to chose which of several theories harmonizes best with facts.

This subject is dealt with in the foregoing detail because of its important relationship to disease in the nasobronchial tract. It has been mentioned previously in this series of reviews that the breathing of cold air and often the effect of cold on the skin in persons sensitive to cold can cause symptoms which have been described in the literature under such terms as vasomotor rhinitis, allergic coryza and asthma, and that heat in persons sensitive to heat, even that generated by physical effort, can produce similar phenomena. Also, reactions of a similar nature can be produced by light in persons sensitive to light. Nasal reactions of this kind are rather common and, if chronic and perennial can cause chronic organic diseases in the nasal membranes and sinuses. The condition does not yield to treatment employed in similar diseases caused by other agents and ought therefore to be diagnosed and taken into consideration when a course of nasal treatment is planned.

In a latter communication, Harris, Lewis and Vaughan [44] reported several interesting cases including those of hemoglobinuria associated with urticaria caused by cold, also urticaria alone caused by cold, and finally, of hemoglobinuria caused by cold that is not associated with urticaria. In a case of hemoglobinuria associated with urticaria caused by cold, they demonstrated the presence of a well known hemolysin which accounted for the hemoglobinuria. They also reported preliminary work which suggests the presence also of a specific dermolysin which they thought might account for the urticarial lesions. They made the important observation that the condition can be transferred passively to the skin of a normal person by the intracutaneous injection of the serum of the person affected—that is, if a small amount of serum of the person sensitive to cold is injected intracutaneously into the skin of a normal person, the application of cold to the area receiving the injection causes the appearance of a typical hive within a period of a few minutes. They found, furthermore, that the attachment of injected sensitizing substance to the skin cells occurs almost immediately and that one reaction permanently desensitizes the area of the injection. These results are almost identical with the studies on the passive transfer of atopic hypersensitiveness. In one of Harris, Lewis and Vaughan's

44. Harris, K. E.; Lewis, T., and Vaughan, J. M.: Haemoglobinuria and Urticaria from Cold Occurring Singly or in Combination: Observations Referring Especially to the Mechanism of Urticaria with Some Remarks upon Raynaud's Disease, Heart **14**:305, 1929.

patients who was sensitive to cold, repeated trial failed to demonstrate the possibility of passive transfer. This is similar to a result that I obtained in the study of a patient sensitive to cold in whom similar efforts at passive transfer gave flatly negative results. These authors refer to a case of cold sensitivity studied by Baumritter in which efforts at passive transfer also gave negative results.

Horton and Brown,[45] in a paper entitled "Systemic Histamine-Like Reactions in Allergy Due to Cold," reported six cases of definitely allergic-like illnesses caused specifically by cold. In two cases, the typical reaction of erythema, itching, redness and swelling limited to the exposed surface could be brought out by immersion of the arm or hand in water at a temperature between 15 and 20 C. Such a reaction would come out from four to six minutes after exposure and would appear best after an exposure to temperatures between 10 and 15 C. These observations harmonize exactly with those made by previous observers both as to time of appearance of the reaction and the optimum temperatures at which the reactions could be elicited. Exposure to a temperature of 20 C. or above gave rise to no reaction whatever. It was observed that the reaction could be retarded by the application of a tourniquet around the arm at the time of exposure, and after removal of the tourniquet, typical constitutional systemic reactions, such as flushing of the face and lowering of blood pressure, would quickly appear. These authors assumed that this indicated a chemical basis for the general disturbance.

These results are similar to several that I obtained in a study of cold sensitiveness but differ from several results obtained in a study of cases of heat sensitiveness. In the latter cases, constitutional reactions could often be brought out by heat applied to the forearm and hand even at a time when tourniquets were applied around the arm so tightly that blood could neither enter nor leave the arm. The reactions were not exaggerated in the least after the removal of the tourniquet. In the latter type of case, one is almost forced to assume that the cause of the constitutional reaction is transmitted through nerve channels. Horton and Brown found that constitutional reactions exactly similar to those produced by cold could be produced by the injection of 0.5 mg. of histamine. The patients were unable to distinguish between the systemic reactions caused by cold and those caused by histamine. They assumed that a physical agent, such as cold, might break down certain protein molecules in the skin or other tissues with the liberation of histamine-like bodies and that these carried into the general circulation might produce general histamine-like effects. They quoted Abel and Kuboto who stated: "What form the agent that kills or injures the cell takes should be

45. Horton, B. T., and Brown, G. E.: Systemic Histamine-Like Reactions in Allergy Due to Cold; A Report of Six Cases, Am. J. M. Sc. **178**:191 (Aug.) 1929.

immaterial. Whether it be heat, mechanical injury, bacteria, or their products, or pharmacologic agents that injure or destroy the cells, in every instance we should expect to find histamine liberated. Nor need the injury necessarily be of so gross a character as to be immediately demonstrable under the microscope."

Horton and Brown are inclined to accept Lewis' view that cold reactions are brought about by the liberation of histamine-like bodies. Their results with therapy are interesting in that they agree with those which I obtained in endeavoring to increase tolerance for cold through treatment with frequent cold baths. They found that a good therapeutic result could be obtained. This differs markedly from the results which I have obtained in many cases of heat sensitiveness through treatment with hot baths. The latter therapy is usually a failure. They reported an important new observation, namely, that in cases of cold sensitiveness benefit can be obtained by small, gradually increasing doses of histamine.

Abstracts from Current Literature

Ear

INNER EAR AND CEREBELLAR CHANGES IN PATHOLOGIC FETAL POSITION. ANTONIN PRECECHTEL, Laryngoscope **39**:421, 1929.

The vestibular organ should be divided, both anatomically and functionally, into two parts; namely, the otolith system and the semicircular canal system. The former system mediates the reactions to position, while the latter responds to the positive or negative acceleration of movement, whether progressive or rotary. It has been shown by various investigators that reflexes due to rotation, such as nystagmus and head deviation, occur in the newly born infant, and by inclining the body in different directions, especially backwards, it is possible to obtain peculiar reflexes of the limbs. These reflexes provoked by the inclination of the body and by progressive movements are of labyrinth origin. As is generally known, the reactions to position are apperception of the vertical and regulation of muscle tonus. The control of muscle tonus has been studied extensively by the school of Magnus and is classed by this school into three groups, namely: (1) controlling of the eyes; (2) reflexes regulating posture; (3) righting reflexes. The first of these does not concern this subject, but the other two jointly control the erect posture and walking of man.

From a careful study of these reflexes together with their effect on posture during the early years of life, it is obvious that the postnatal static and locomotive functions are considerably influenced by the vestibular apparatus. The author believes that it is only logical that one ask whether the typical position of the fetus, and possibly even the attitude of the fetus, is likewise controlled by the vestibular apparatus, in view of the fact that the labyrinth reflexes have been detected both in fetuses and immediately after birth. Various theories have been advanced by Aristotle, Simpson, Kristeller, Griffith and others as to what controls the position of the fetus toward the end of pregnancy, but these, the author believes, have all been disproved. On the other hand, he asserts that none of the facts contradict the explanation of the mentioned position by a static function of the fetus itself, a function reflectorily controlled by the organ which has been proved to serve, in conjunction with other reflexes, the purpose of orientation in space. This view offers a uniform regulation of movements and positions in all the periods of life and is compatible with the modern physiologic theory of the kind of regulation, and is also supported by the fact that the vestibular organ is developed at a remarkably early period. Bechtierew has discovered that the vestibular nerve myelinizes long before the cochlear and other sensitive nerves. It has been found by Flechsig and His in examining a 16 cm. fetus that the branch of the vestibular nerve going to the superior semicircular canal was myelinized as far as the ganglion of Scarpa, and the ramifications of the macula sacculi and utriculi were also myelinized at this stage. It was found in a fetus from 18 to 19 cm. long that the cupulae with hairlets were developed, and Minkowski stated that the maculae develop when the fetus is only two months old. Consequently, tonic influence is exercised early on the various muscle groups. The movements of the labyrinth fluid, which is influenced by gravity, prove a specific stimulus for the vestibular nerve. In this connection it may be stated that the period in which the myelinization of the vestibular nerve is accomplished covers also the stabilization of the normal position of the fetus at the end of pregnancy.

The author attempted to support this static theory of fetal positions still further by clinically examining such cases as showed abnormal position during the fetal period. A considerable number of such cases appear at otolaryngologic clinics, the

patients' complaints falling within the sphere of phoniatry. In the cases studied, the author found, besides an anomalous position during the fetal period, static disturbances in the early postnatal years, as evidenced by a retarded power of sitting, standing and walking. This syndrome is sometimes attended by a speech defect, known as audimutitas, and caused by changes in the cerebellum in the region of the lingula. The lingula is one of the structures of the basal stage of the cerebellum, to which the vestibulocerebellar paths directly converge, and it plays a certain part in the movement of the vocal cords.

The author studied six cases with a history of pathologic fetal positions. In two of them histologic changes were found in the inner ear and in the central nervous system; in one the changes were in the central nervous system only; while in the remaining three the brain was not examined, but in two of them changes in the inner ear were found, thus making a total of five cases with pathologic changes, out of the six cases under study. The peripheral changes consisted in a heavy hypotonic degeneration of the neuro-epithelium in the cochlea and in the vestibular apparatus. In addition, one case presented an extensive primary disturbance of the development of the inner and of the middle ear. The changes were either unilateral or bilateral. The central changes affected the medulla, the midbrain or the cerebellum.

The speech disturbances which are frequently associated with the syndrome may be divided into two groups: (1) speech disturbances of those who are hard of hearing and (2) audiomutism. The peripheral changes explain the disturbances of speech based on decreased hearing, while audiomutism is related to functional disturbances of the cerebellum. The changes in the lingula are especially suggestive in these speech disorders, owing to its relation to the movements of the vocal cords.

SNAPP, Grand Rapids, Mich.

THE USE OF AYER'S SPINAL MANOMETER IN LATERAL VENOUS SINUS PHLEBITIS. GEORGE O. CUMMINGS, Laryngoscope **39**:455, 1929.

The use of this instrument in cases of bilateral acute mastoiditis, complicated by lateral venous sinus phlebitis, have proved extremely valuable in determining which sinus is involved. In cases of bilateral acute mastoiditis in which symptoms of phlebitis of the lateral sinus occur, it is difficult and often impossible to determine clinically which side is affected, without operating and actually inspecting the sinus. In such instances, the author does an ordinary spinal puncture, and after the needle is in place, an Ayer's manometer is attached and the reading taken. Compression is then made on one jugular vein and the reading noted, and the procedure repeated on the jugular vein of the opposite side. If there is a decided difference in the readings of the two sides, it is concluded that the side which reveals the least rise in millimeters of water is partially occluded, thus suggesting mural thrombus. In case the sinus on one side is totally occluded, compression of the jugular vein of that side will cause no change in the fluid level in the instrument, while if the other sinus is normal, compression of the jugular on the normal side will cause the fluid to rise sharply to around 360 mm. of water, thus suggesting occlusion of the opposite side.

SNAPP, Grand Rapids, Mich.

Pharynx

NERVE BLOCK IN AMYGDALECTOMY. HOMER A. TROTTER, Ann. Otol. Rhin. &. Laryng. **38**:376, 1929.

Postoperative pain and discomfort following enucleation of the tonsils is extremely distressing and its elimination would be highly appreciated by both patient and surgeon. The author finds that postoperative pain can be eliminated by nerve

block into the nerve supply of the tonsil if proper technic is administered. He reports having operated on 126 patients by this method with very satisfactory results.

The nerve supply to the tonsil is from the posterior palatine and the rami of the glossopharyngeal nerve. These nerve trunks converge and run side by side as they approach the tonsil and are in immediate relation with the tonsillopharyngeus muscle. This muscle bundle is attached to the groove at the junction of the middle and lower third of the tonsil. It holds the tonsil in position and is accompanied by the nerve, blood and lymphatic supply to the tonsil. Injection of an anesthetic solution into the tonsillopharyngeus area will produce a nerve block because at this point both nerves run parallel. In no other location can nerve block be accomplished by a single injection.

Quinine and urea hydrochloride solution is the author's choice of anesthetic, as it produces a much more prolonged anesthesia than procaine hydrochloride, the effects lasting in some cases as long as eight days. The author's technic consists in placing the patient in the upright position and painting the throat and tongue with cocaine hydrochloride (flake). No preoperative narcotic is administered. A specially built nonflexible cannula needle, 23 gage, with three fourths of an inch penetrating depth, is required. One cubic centimeter of 0.5 per cent solution of quinine and urea hydrochloride is injected back of the capsule of either tonsil in the region of the tonsillopharyngeus muscle, the needle piercing the lower one third of the palatoglossus muscle near its median line. The needle then enters a space occupied by areolar tissue and comes in contact with the capsule of the tonsil. The edge of the capsule is followed until resistance is met, which resistance is the tonsillopharyngeus muscle. Injection is made into this muscle bundle to produce nerve block. Complete anesthesia is obtained after fifteen minutes, and the tonsils are then enucleated by the dissection and snare method.

CARL F. SNAPP, Grand Rapids, Mich.

Nose

ANAPHYLAXIS AND ALLERGY IN RHINOLOGY. FRANCIS P. EMERSON, Ann. Otol. Rhin. & Laryng. **38**:313, 1929.

Clinically, the symptomatology of anaphylaxis is the same as that of allergy in every detail, but there is an essential difference due to underlying causes, which creates a problem for the biologist rather than for the clinician. The clinical literature has dealt mainly with the nonbacterial type of protein sensitization, probably because it was easier to discover the foreign protein and to establish immunity by the exhibition of small doses or to withdraw the offending food entirely. Theoretically, it is true that anaphylaxis may be due to the failure of a specific antigen to elaborate amino-acids sufficient to render the ingested foreign protein harmless. Then, too, there is an inherited protein susceptibility, as has been shown in various cases. The author concludes that hypersensitiveness, which may be seasonable or all the year round, being manifest at one time as hay-fever and at another as asthma or vasomotor rhinitis, is simply unstable cell equilibrium from some unknown etiology. The endocrine balance is also an important factor here, there being a previous lesion of the endocrine sympathetic system in cases of anaphylactic shock. If the endocrine system is normal, it resists toxicity, but if it is out of balance it may be easily affected.

The author has observed that there is a chronic maxillary sinusitis with fibrosis and infiltration, but without pus, in all his cases of asthma. Some fifteen patients have been studied, all of whom were operated on by the Caldwell-Luc method. All the thickened, fibrosed lining membrane was completely stripped out in each instance. Simply to establish drainage and ventilation in such cases is not sufficient. The follow-up treatment is especially important, all patients being kept under close observation for at least a year. The results in the author's series are remarkably good, all cases being greatly improved or cured. The observations, however, have

extended only over a period of from one to two years. The author has also found that many patients with hay-fever have the same condition in the maxillary sinuses as is found in the cases of asthma.

SNAPP, Grand Rapids, Mich.

THE COINCIDENCE OF MENINGEAL CALCIFICATION AND NASAL PATHOLOGY. JAMES A. BABBITT, Ann. Otol. Rhin. & Laryng. **38**:320, 1929.

Calcification of the meninges is less common in man than in the lower animals, but still it cannot be considered rare. It formerly was thought to be a finality in itself, whose completed process was of little pathologic menace, but more recently there has been noted a definite coincidence between its occurrence and that of deep infection of the sinuses. The atypical neuralgias are frequently associated with meningeal calcification, and the author believes that the interest of the subject to the laryngologist lies in the possible establishment of sufficient connection to furnish added diagnostic interpretation of deep perisinusitis, and in so doing to afford another link in the baffling study of atypical neuralgias.

There is little presented in the literature indicating pathologic coincidence or relationship of meningeal calcification to pathologic process in the sinuses. However, it has been cited by several writers. Hirtz reports a series of 144 cases studied in relationship to the pineal gland, and of this series 56 per cent revealed calcification; but calcification was not found without associated evidence of deep perisinusitis. The falx easily assumes first place as the common site of the condition, with the areas in the dura alongside the superior longitudinal sinus, and the tentorium being next in frequency. All the areas of the convexity may be affected. The dura may be involved alone, or together with the other membranes. A congestion of the vessels in the involved area is usually present, and adhesions are frequently observed. It has been stated by Weber that ossifications of the dura do not belong to the class of genuine tumors but to inflammations. The author and others believe that these osteitis changes are thrown out as a defense in deep perisinus areas and the difficult point is to establish any link of continuity between this process in the surrounding bone tissue and the calcification process in the meningeal structure itself.

The author observed six cases, all of which presented striking phases of neuralgic pain-complex. Each of these cases presented intranasal pathologic process of a degree justifying surgical intervention. Of these six, four presented calcification at the base of the brain around the pituitary area, and two higher about the longitudinal sinus. These cases are not often recognized, and the author pleads for a more conclusive roentgen study and closer observation.

CARL F. SNAPP, Grand Rapids, Mich.

Miscellaneous

PERFORATION OF THE ESOPHAGUS WITH REPORT OF SIX CASES. EDWARD KING, Ann. Otol. Rhin. & Laryng. **38**:351, 1929.

In reporting six cases of perforation of the esophagus, the author is very emphatic in warning against the pushing into the esophagus blindly to dislodge a foreign body. In all cases of sharp foreign bodies there is great danger of perforation, so that extreme care must be exercised even with the esophagoscope. In four of the six cases reported the perforation was caused by chicken bones, in one by a thumb tack which had been accidently swallowed by a child, and in one case the perforation resulted from a metal bougie which was being passed without a guide in an attempt to dilate a lye stricture. In two of the cases the perforation occurred in the thoracic region, while in four it was in the cervical. In three of the four cases in which the perforation was caused by a chicken bone, stomach tubes had been passed by the physician in an attempt to dislodge the bone. There

was one death in the author's series, this being caused by a marked periesophagitis with extensive involvement of the mediastinum together with abscess formation.

It is evident from the author's series that perforations occur in the esophagus without the formation of pus. Esophagitis and periesophagitis appear; the patient has a reaction, and the infection subsides. This happens in the thoracic as well as in the cervic region. Perforation at once exposes the mediastinum to infection, consequently surgical drainage is the only form of treatment in such cases and this should be done immediately if it is to be of much value. Opinions differ as to the indications for external surgical intervention. In considering external surgical measures, pain is usually the first symptom, followed rapidly by fever, rapid pulse and difficulty in swallowing. Swelling of the tissues and emphysema also occur. The emphysema may be present in a slight degree and remain unrecognized unless a roentgen examination is made. With these signs present, together with a high white cell count, the author believes there is every reason for opening the neck. The infection first establishes itself in the loose periesophageal tissues, and from there spreads out into the fascial planes of the neck, descending between the sheaths into the mediastinum. To open the neck and drain this area is not a difficult procedure; it has been referred to as a prophylactic mediastinotomy.

CARL F. SNAPP, Grand Rapids, Mich.

THE CAUSES OF RETROBULBAR NEURITIS. RICHARD SCHEERER, Klin. Monatsbl. f. Augenh. **83**:164 (Aug.-Sept.) 1929.

Scheerer has collected and studied his material at the Tübingen University Eye Clinic for the years 1921 to 1928.

Among 64,200 patients, he has seen 203 cases of acute and chronic retrobulbar neuritis. Fifty-four per cent occurred in men, and 46 per cent in women. Of these cases, 16 per cent were of different recognizable causes, such as tobacco, alcohol, nephritis, arteriosclerosis, syphilis, brain tumors, encephalitis, etc. Seventy-one and five-tenths per cent, or 145 cases, occurred in certain and likely cases of multiple sclerosis; 1.5 per cent, or three cases, occurred in obvious nasal sinusitis and 11 per cent, or twenty-three cases, were of undetermined cause.

When the material seen in consultation at other clinics was deducted, there remained forty-six positive and eight probable cases of multiple sclerosis with retrobulbar neuritis, three cases due to nasal sinus disease and twenty-three cases of unknown origin.

Altogether, the author saw 214 cases of multiple sclerosis about equally distributed between men and women. Of these, 134, or 63 per cent, had fresh or subsiding retrobulbar neuritis.

The author divided his cases according to the decades of life, and found that 33 per cent occurred in the first two, 33 per cent in the third and fourth decades, and 20 per cent after the age of 40. He found that the decline in the cases of retrobulbar neuritis after the age of 40 was due to the death of many of the patients.

He divided the cases of multiple sclerosis into light and severe forms. The light cases were those with slightly disturbed reflexes. The suspicious cases were those which showed only a few disturbances. There were, in all, eleven suspicious cases. Added to the light cases were those having transitory disturbances of eye muscle, of which 15 per cent showed these disturbances on the same side as the optic neuritis. The severe cases were classed as those showing general intention tremor, ataxia, disturbances of general sensation and disturbances of the bladder.

Forty-nine per cent of the cases of retrobulbar neuritis were unilateral, and 51 per cent were bilateral, similar, as he says, to what Uhthoff found.

Only four cases had subjective flashes of light, but more frequently were present headache and pains about the eyes.

Five patients were completely blinded at the beginning, one of which remained so for six days, two for nine days, and two for fourteen days. One of the latter regained only five-twelfths vision, but all the others regained full visual acuity.

Scheerer stated that the prognosis for the return of vision in retrobulbar neuritis due to multiple sclerosis was good. He stated that only seldom was there a true, severe disturbance of sight, although, naturally, some loss, especially for colors, remained. Only one eye in all the cases seen stayed blind.

The observations on the visual field showed unilateral central scotoma in 34 per cent of the 214 cases of multiple sclerosis, bilateral scotoma in 32 per cent, paracentral 6 per cent, peripheral narrowing in 8 per cent, and the field was normal in spite of obvious optic atrophy in 20 per cent.

The fundus showed temporal atrophy in 34 per cent, total atrophy in 14 per cent, faded appearance of the papilla in 6 per cent and normal observations in 46 per cent. In the latter group, 20 per cent showed severe visual disturbance, in which the results of the fundus examination were normal. There were eleven recurrent cases.

Scheerer states that there were only three cases of certain nasal sinus disease in his entire experience; namely, 1.5 per cent of the cases of retrobulbar neuritis showed certain nasal sinus disease. He stated, however, that there were five other cases in which the possibilities of a nasal cause existed, but two of these developed later into multiple sclerosis. The condition in one was diagnosed as tobacco-alcohol intoxication, and in two it remained undiagnosed. The patients in three other cases, in spite of normal observations on the nasal sinus were operated on. Two of these showed improvement, and one became worse.

In discussing the twenty-three cases in which the condition was not diagnosed or 11 per cent of the material, three of these were the cases in which operation was done, two followed an attack of grip, one was of dental origin, one was hereditary neuritis, one was a skull fracture, two were neurologic cases with nystagmus, and thirteen cases gave no clue as to the cause. All the patients in the latter group recovered, as did those in the other acute cases. Sometimes, the duration of acute symptoms varied from less than a year to six years.

In discussing the high figure of 30 per cent of cases in which the condition was not diagnosed, one might argue, according to Scheerer, that these belonged to the group of nasal sinus etiology, but he felt this was not right as all of the patients in these cases recovered under conservative treatment without any operative procedure on the nasal sinus. He said that had these been operated on, the fortunate outcome would have been assigned to a nasal sinus etiology. He felt that the only proper way was to wait to see whether the general symptoms of multiple sclerosis appeared or not. This, he has done in forty-six cases of retrobulbar neuritis due to multiple sclerosis. In some cases, it was sixteen years before the other signs of multiple sclerosis appeared.

In discussing the points of view of the rhinologists in the cases of retrobulbar neuritis, he states that the most delicate clouding in the x-ray pictures and insignificant changes in the mucosa have been regarded by rhinologists as the establishment of a sinus disease as an etiologic factor. He said he thought it would be advisable for the adherents to the rhinologist's theory to see whether multiple sclerosis developed later. Besides, he felt that rhinologists should study the eyes in all of their nasal sinus cases, and if an optic neuritis of nasal origin was frequent, it would be found in these cases. Furthermore, he felt that though latent nasal sinus disease is frequent, cases of retrobulbar neuritis are extraordinarily rare.

He felt that because favorable results were obtained after nasal sinus surgery, it did not prove that these cases were of nasal origin. Furthermore, he mentioned that there were some cases of blindness following nasal sinus surgery in which the eyes had been normal before operation.

In discussing the large number of cases of multiple sclerosis seen by him, Scheerer said there is a possibility of geographical difference, as in Wittenberg, multiple sclerosis was frequent.

His treatment of retrobulbar neuritis in the cases of multiple sclerosis consisted of 0.15 Gm. neoarsphenamine weekly, besides the usual general treatment. Often, there was a noticeable improvement in the vision the day after the injection of neoarsphenamine.

Of the fifty-four cases of retrobulbar optic neuritis which he personally controlled, 82 per cent of the patients recovered, and 18 per cent were unimproved.

He stated that nasal sinus disease cannot play an important rôle in the cause of retrobulbar neuritis, as only 1.5 per cent of his cases were due to nasal sinus disease.

H. H. Vail, Cincinnati.

Society Transactions

AMERICAN LARYNGOLOGICAL ASSOCIATION

C. J. IMPERATORI, M.D., *Abstract Editor*

Fifty-First Annual Congress, Atlantic City, N. J., May 20-22, 1929

CHARLES W. RICHARDSON, M.D., *President, in the Chair*

PRESIDENTIAL ADDRESS. DR. CHARLES W. RICHARDSON, Washington, D. C.

This is the commencement of the second half century of the American Laryngological Association, and I regret my inability to draw a picture as to what will transpire during the next half century, but there will be many problems that will be presented for elucidation. The history of the Laryngological Association is divided into three periods: first, that of the discussion of the larynx and pharynx; second, that of the discussion of the nose and its sinuses and their adnexa, and third, the period of endoscopy. Attention is directed to the importance of food as a factor in health or its deficiency as a factor in disease. Watson considers the science of nutrition as one of the basic sciences and makes an apt quotation from Dr. McCarrison's work on "Studies of Deficiency Diet." After stressing this fact, attention is called to an important article of food which in all probability will not be considered by the members of the symposium, and that is sugar. The influence of the excess of sugar in promotion of diseases of the upper air tract should be given more attention. I feel that the junior specialist is neglecting his knowledge of clinical medicine, and his attention must be directed to overcome this deficiency.

CONGENITAL LARYNGEAL STRIDOR. DR. FREDERICK T. HILL, Waterville, Maine.

In reviewing the cases reported in the literature, considerable variation in the exact laryngeal picture is noted. In a case herein reported, a peculiar twisting movement of the epiglottis as it was sucked downward during inspiration is described. These variations, as well as the different results in the experiments of Thomson and Turner, point to there being some essential variation or malformation in the structures of the laryngeal aperture themselves, as the primary cause of the stridor, as against the "central theory." These structures have a different histology predisposing to flaccidity, and are of different embryologic origin, from that of the rest of the larynx. Being found only in mammals and varying somewhat in different species, they may be considered as a later acquisition, and consequently more subject to congenital malformation. Hence it would seem that there are grounds for considering that the inherent cause of the stridor lies in the structure of the laryngeal aperture.

DISCUSSION

DR. J. H. BRYAN, Washington, D. C.: I wish to cite the history of a patient with laryngeal stridor. This child was badly nourished, born of nervous parents. When first seen, he was in a state of quiescence. There was no evidence of stridor. On the slightest excitement, the stridor would begin. Cohen described the condition as a falling epiglottis, which he relieved by passing his finger down into the larynx and pulling the epiglottis forward. I was called suddenly to see the child, and there was a marked stridulous breathing. I put my finger down into the larynx and attempted to pull the epiglottis forward, as Dr. Cohen had suggested. I found it was caught between the epiglottic folds. After that, the child progressed well. I sent the child to Dr. Cohen for a verification of the diagnosis and for future treatment. He did not agree and considered the condition laryn-

gismus stridulus due to rickets. As the child grew and the larynx increased in size, the stridor disappeared.

DR. D. B. DELAVAN, New York: To cite a case, a new-born infant, particularly healthy and well, on the second day developed marked stridor. I had read that remarkable contribution to laryngology by the late Dr. J. Solis Cohen, who explained that such laryngeal stridor was apt to be due to falling of the epiglottis. Remembering that, I had with me a small celluloid spatula, curved at the end, something in the shape of a spoon. Excellent laryngoscopic examination of the child was made. The epiglottis was flaccid and was inclined to fall on the top of the larynx. I used the spatula to draw the tongue forward and downward, and the stridor was at once relieved temporarily. Whenever an attack came on, this simple method of treatment was used; in the course of four or five days the condition began to disappear, and finally vanished. The patient is today middle aged, a fine example of physical and intellectual development. He has never since had laryngeal trouble.

The thesis on the subject by the late Dr. J. E. Newcomb, was a valuable contribution.

DR. H. L. SWAIN, New Haven, Conn.: Germane to the subject before us, the difficulty of breathing in the new-born, which sometimes apparently is due to bulbar paralysis or difficulty originating in that area, results in the infants not breathing. There is no particular obstruction to breathing, but they do not breathe. The details of a case such as this were recited, in which the attempt was made with the bronchoscope to keep the epiglottis from falling tight into the glottis. This was done but it did not save the child's life. Artificial breathing was maintained for a long time.

DR. M. C. MYERSON, New York: A majority, if not all, of the larynges of these infants are the same in contour and anatomy, but because of the emotional make-up, or may I say because of something in its heredity, one child reacts more severely than another, as far as its respiratory activity is concerned, and instead of a normal inspiratory effort that child has an exaggerated inspiratory effort when it is emotionally excited, or excited by any outside stimulus. It is this factor that is usually lost sight of.

CHONDROMA OF THE LARYNX. DR. LOUIS H. CLERF, Philadelphia.

The occurrence of this type of tumor is relatively infrequent. Diagnosis is often difficult. Individual experience is necessarily limited. The tumor often has its beginning in the subglottic region and cannot be seen by mirror laryngoscopy, especially during phonation. Immobility of one vocal cord is often the first evidence of laryngeal disease. There is a question of malignant disease. In the case of chondroma of the larynx reported, when the patient was first examined, bilateral involvement of the vocal cords was observed with no evidence of tumor. At examination four years later, there was marked dyspnea with dysphagia. Treatment was instituted.

DISCUSSION

DR. W. B. CHAMBERLIN, Cleveland: I had two experiences in each of which I made a preliminary diagnosis of chondroma of the larynx. The first was in a child about 4 years of age. I palpated this tumor with a probe and thought that it must be a chondroma; it was so dense and firm and hard. It proved to be a cyst. The other was in an adult, the tumor being the size of a large hazelnut. I thought from the consistency of this tumor that it must be a chondroma of the larynx. It proved to be a large tuberculoma with a predominant amount of fibrous tissue.

DR. H. L. SWAIN: (A demonstration was given with a pencil sketch.) The tumor on the right proved to be a cancer; the one on the left, a hard growth. It was as hard as Dr. Chamberlin said the tumor in his case was. I did exactly as he did, to prove my diagnosis, and plunged a knife in. The tumor did not collapse

but a little fluid came out. It was a thick-walled cyst, and as it was so large it did not collapse enough to give the patient sufficient air space. Dr. Verdi, of New Haven, shelled the cyst out by an external incision entirely free from any break into the interior of the larynx. The patient is well.

DR. R. C. LYNCH, New Orleans: I wish to talk about Dr. Clerf's paper for although the case does not belong to the group of cases of chondroma, the symptoms and occurrences parallel exactly the description given by Dr. Clerf. I saw this patient four years ago. He had fixation of one cord, lagging of motion in the other, difficulty in breathing, and a hard tumor below the cord. An x-ray picture showed a dense tumor apparently occupying the posterior surface of the larynx, subglottically. We did a median thyrotomy and found the tumor quite hard; 50 mg. of radium was put in for eight hours. To our surprise, the tumor melted away. The function of the cord came back, so that the patient was able to breathe fairly well. About a year or fifteen months later, obstruction reappeared, fixation of the cord reoccurred and tracheotomy was necessary.

Last year a hard, protruding tumor appeared around the tracheotomy wound. When cut into, it looked like an Irish potato, and to our great surprise, microscopic examination showed a mixed tumor of the parotid type. This shows another one of the differentiations that must be made between chondroma and mixed tumors of this type.

ENDOTHELIOMA OF THE LARYNX. DR. ROBERT CLYDE LYNCH, New Orleans.

Seven patients with hemangio-endothelioma of the larynx have been operated on during the last twelve years. All of the cases were in men between 30 and 45 years of age. The patient's history throws no light on the etiology. All the growths developed as hemorrhoidal-like tags from a point half way between the vocal process and the posterior extremity of the arytenoid on its upper surface. There is no interference in the tone of voice but a feeling of laryngeal discomfort is noted without pain, either local or reflex. Pathologic points bearing on this condition are discussed. Removal by suspension, dissection and diathermy coagulation have given the best results, the patients in all cases recovering with good functional tone to the voice and no recurrence of tumor. In the last case, a year has elapsed since the operation.

Microscopic slides, both low and high power magnification, show the lawless endothelial proliferation characteristic of the tumor.

DISCUSSION

DR. H. G. TOBEY, Boston: I should like to corroborate Dr. Lynch's statement as to the rarity of these tumors, and I am glad to see that he used diathermy. I would rather, perhaps, call it electrosurgery. Angiomas of the larynx are exceedingly rare. I have had experience with three cases of hemangio-endothelioma of the nasal septums: in one the patient responded successfully to electrosurgery, in the other two, in which the patients were treated before I began to use it, complete removal of the septum and the tumors was necessary before recurrences could be stopped.

DR. R. C. LYNCH: I should like to add that the endotheliomas that occur in the soft palate, tonsil and nasal pharynx seem to be, from a prognostic standpoint, entirely different from those tumors which I have observed in the larynx. Second, I should like to say that it is possible, of course, for a hemangio-endothelioma to grow from any part of the body. They grow from nerve tissue, in cartilage, in fact, they grow anywhere. That, I think, ought to be mentioned, and also that the cavernous group is rare apparently and is supposed to be congenital.

I wanted to get your minds fixed on that point, that none of these tumors were congenital, that none of them had the usual dark blue or purple wine spot birthmark appearance, but that they looked more like a hemorrhoidal tag, and grew evidently from the depth of the tissue.

PRESENTATION OF PATIENTS, INSTRUMENTS AND REPORTS. DR. H. G. TOBEY, Boston.

Dr. Tobey presented a retractor for doing a radical antrum operation. It has a narrow tip with a raised point, in a more or less triangular shape, curved on the under side. The instrument fits under the periosteum, raises the cheek, keeps it out and prevents it from dropping together. The advantage of using this instrument is that it takes only one retractor. If necessary, the anesthetist can hold it easily by this handle. It gives a good view into the antrum without the cheek falling down over the retractor.

DR. C. W. RICHARDSON: Dr. Charlton, who has always been very courteous to us whenever we have come to Atlantic City, although not a member of the organization, has an interesting case which he would like to present, and if there is no objection, I shall consider that unanimous consent is given and Dr. Charlton may bring his patient forward.

PRESENTATION OF PATIENT. DR. C. C. CHARLTON, Atlantic City, N. J.

This is a case of paralysis of the left vocal cord due to an aneurysm. The interesting part of it is that with the patient's head in this position (indicating) he can talk fairly well. If his neck is straightened out or turned to the other side, there is almost complete loss of voice.

The man is 44 years of age, and is a plumber. He does not give a history of having had any lesions of syphilis. The Wassermann reaction is four plus. It is an inherited condition.

DR. CHAMBERLIN: Is the paralysis unilateral or bilateral?

DR. CHARLTON: Unilateral.

DISCUSSION

DR. L. W. DEAN, Iowa City: I should like to ask Dr. Charlton if when this patient is talking and then turns his head from one side to the other, the voice will not gradually disappear. I have noted that. Just turn it slowly, instead of rapidly, while he is counting. (The patient turned his head slowly from right to left, counting as he turned. His voice gradually disappeared.)

DR. D. B. DELAVAN presented a postnasal syringe devised by Dr. Thomas Rushmore French.

SYMPOSIUM

VITAMINS IN HUMAN AND ANIMAL NUTRITION. DR. R. ADAMS DUTCHER, Pennsylvania.

A general discussion was given on the development of the vitamin hypothesis with a description of research methods and their evolution. Vitamins A, B, C, D, E, F and G were discussed from the standpoints of occurrence, properties, isolation, purification and function. Recent developments in research relating to vitamins, ultraviolet light, food activation, vitamin destruction and vitamin synthesis were described.

DIET IN CONNECTION WITH DISEASES OF THE LYMPHATIC SYSTEM, ESPECIALLY OF THE UPPER AIR TRACT. DR. ROY A. BARLOW, Madison.

As there is no literature dealing with this subject, it was necessary to conduct my own laboratory experiments. Albino rats were used and were fed various diets deficient in vitamin A, B, C and D. Microscopic sections showed no changes in the lymphatics. Animals on starvation diet and those fed on excessive fat diet showed no changes. I would conclude from these experiments that diet has no direct effect on the lymphatic system. Further investigation over longer periods of time might possibly alter these observations.

THE RELATION OF DEFICIENCY DIET TO DISEASES OF THE SINUSES. DR. LEE WALLACE DEAN, St. Louis.

Nasal sinus infection may be produced in rats by a diet deficient in vitamin A. Correction of diet in early stages of sinus infection produces satisfactory results. In later stages, it does not. Results secured in infection by a diet rich in vitamin A were satisfactory. When sinus infection is present in the human being, vitamin A diets are important therapeutic procedures.

DEFICIENCY DIET IN RELATION TO THE SKELETON, ESPECIALLY IN CONNECTION WITH THE BONE DISEASES OF THE HEAD. DR. BURT R. SHURLY, Detroit.

1. The structure, stability and growth of the bones of the head are dependent on the supply of vitamins A, C and D, particularly with a determined ration of calcium and potassium.

2. The prevention of rickets is a prenatal problem together with the diet of the nursing mother. The value of cod liver oil, milk, eggs and leafy vegetables needs wider application.

3. Irradiated ergosterol is equivalent to vitamin D and offers a concentrated form of preventive treatment for rickets.

4. Simple hyperplastic goiter with the attending growth problems in the Detroit public schools has diminished in five years from 42 to 7 per cent with the use of iodine.

5. Many skeletal defects in our school children, decayed and crowded teeth, small sinuses, high arched palate, bony asymmetry and deflections, congenital thyroid, may be modified by prenatal care with special attention to a supply of iodine, raw milk, egg yolk, leafy vegetables and orange and tomato juice with irradiated ergosterol to the pregnant and nursing mother.

THE NECESSITY FOR A SCIENTIFIC INVESTIGATION OF THE CAUSES OF THE PREVALENT CONDITIONS OF HYPERTROPHY OF THE LYMPHOID ELEMENTS OF THE PHARYNX. DR. D. BRYSON DELAVAN, New York.

The Journal of the American Medical Association says editorially, "A recent Government report estimates that about one third of all operations since 1924, among the American urban population, were for the removal of tonsils or adenoids. The urgent need for comprehensive statistical studies of the results is apparent."

The study of the tonsil has been attempted for many years, but often in a desultory and speculative manner with little practical result excepting as shown in such pioneer work as that of Dr. French, Dr. Shambaugh and a few others. What is now demanded is the study of the prevention of the underlying conditions which have given rise to such a wide surgical intervention.

It is therefore urgently recommended that the same principles be adopted for these purposes of investigation as are being used in the solving of other great pathologic problems, and in particular that the investigation of the basic causes of hypertrophy of the lymphoid elements of the pharynx—hereditary, hygienic, dietetic, climatic and otherwise—be undertaken by a group of highly trained research workers; that for this purpose adequate financial support be supplied, suitable laboratory facilities provided, and every possible aid given by the medical profession at large.

For the successful management of this project there should be a central responsible body, representative of the best talent of the country, the high standing of whose members would impart dignity, intelligence and energetic effort to the work. For the fulfillment of this, no organization could exert greater influence in bringing about the existence and the success of such a movement than the American Laryngological Association.

DISCUSSION

DR. J. F. BARNHILL, Indianapolis: Dr. Daniel's work has been mentioned here by Dr. Dean, and although it is first class, her teaching has been confusing. She

once stated before the American Academy, as I understood her, that lack of vitamin would produce from the few bacteria which in themselves would produce foci of disease. Fearing that I misunderstood her, I asked this question: "Can you create bacteria from the few in the living body by a lack of a given vitamin?" and she answered, "Yes."

This was refuted so successfully by Dr. Dean just now that further discussion is unnecessary. But over and over again members have gone away from our meetings, after hearing papers presented on the question of vitamins, apparently believing that sinus diseases may be caused by lack of vitamins, and not by bacteria, which are really known to be the cause. As I see it, and as I believe it has been brought out clearly today, the lack of vitamins in the system merely predisposes the subject to the action of bacteria. It is the bacteria after all that actually cause the sinus disease.

I should like Professor Dutcher to tell what really happens to people who eat nothing but meat and to those who eat nothing but vegetables. There are both classes. In South America, until recently, dwellers on the Pampas ate almost nothing but meat. In England, and in fact in all European countries, up until comparatively recently, almost all the people were meat-eaters. Eskimos eat nothing but fat.

What happened to those people? Did their teeth become affected early, and did they suffer more from sinus conditions than we? I am asking this not in a captious way but for information, for those who have been studying these questions carefully should be able to tell us. What is happening to the Eskimo, for instance, who eats nothing but fat, and what happened to Englishmen of the time of King Henry the Eighth who ate almost nothing but meat?

DR. W. B. CHAMBERLIN: In reply to Dr. Barnhill's query, a statement of Stefansson's occurs to me in relation to his life in the Arctic among the Eskimos. His explanation of how he could live for a long time on nothing but a meat diet was that the meat was not in a cooked state but largely in a raw or semiraw condition. Stefansson, of course, demonstrated conclusively that life could be carried on there on the food which the country afforded and, above all, the subjects remained in perfect health on nothing but a meat diet.

DR. H. L. SWAIN: With regard to the remarks just made by Dr. Barnhill about the bacteria, Stephen Maher of New Haven has studied the bacillus of tuberculosis from various angles. He has found that acid-fast bacilli can be made nonacid-fast, and he can take those nonacid-fast bacilli and make them acid-fast again by varying the culture medium, that is to say, the diet and the conditions by which the incubation in the laboratory is allowed to take place.

The idea that if one changes the diet in a person a germ which was innocuous and latent can be made to become a formidable foe by changing its environment seems feasible.

I should like to emphasize Dr. Delavan's remarks on the question of why we do not do something else besides cut out tonsils and adenoids. Why do not we try to make a race that grows up without having these tissues enlarge?

DR. J. D. KERNAN, New York: I am going to say a word or two about the meat-eaters because perhaps I am one of the few here who have had an opportunity to observe the Eskimo on a purely meat diet and the Eskimo as corrupted by the white man's diet.

I had an opportunity about thirty years ago in Greenland to see the North Greenland Eskimos, and at that time they were on a purely meat diet, and all the children, and the hunters, too, were in the most vigorous health. It was a curious thing to me at that time to see little babies less than a year old being handed chunks of raw meat to break their teeth on. And in addition to that, the babies were suckled very late. I saw children 3 and 4 years old still nursing.

During the past summer, I was in Labrador where, due to the whalers, sealers and the fishermen, the game has gradually been killed. Seal, walrus, even, and the cod and salmon are going. All the caribou have disappeared from the interior of Labrador, and the Indians and Eskimos are now being fed on sugar, flour, canned

goods, and so on, introduced by the Hudson Bay Company. The result is that the native population is rapidly disappearing.

When the influenza epidemic struck the coast of Labrador in 1919, in one of the Eskimo villages where there were 400 people, 350 of them died. Their resistance was absolutely gone, probably the effect of their diet.

Another curious thing is that at the time I was there, there had been no adenoid and tonsil trouble among the children. Now Dr. Grenfell has one or two laryngologists traveling up and down the coast removing tonsils and adenoids. That is probably the effect of diet.

I have often thought that adenoids and tonsil trouble might be caused by the continual insult to the nasal and throat mucous membrane offered by the gas and dust of civilization but, of course, in Labrador, there is still no gas and dust from civilization.

DR. R. ADAMS DUTCHER: I have been asked, I think it was by Dr. Taylor, where he might recommend that the laity obtain some information in a semi-technical way on nutrition and dietetics, particularly the vitamin phase, and I thought you might be interested to know that for the physician who is interested in the more technical phases of the work, I consider McCollum's "Newer Knowledge of Nutrition," published by the Macmillan Company, a good general book.

Dr. H. C. Sherman, collaborating with Miss Sybil Smith, has a monograph, entitled "Vitamins," published by the American Chemical Society, the new edition of which ought to be out within the next few months. I think that this last edition is published by the Chemical Catalog Company.

But for the laity I can recommend no better publications (and it reads like a novel) than the publications of the Chemical Foundation, and I would recommend them to every physician, as well, to read, if they have not already done so.

Are you familiar with the publications of the Chemical Foundation, 85 Beaver Street, New York, financed by Mr. Garvan, who has been financing the publication of popular books, "Chemistry in Relation to Medicine," "Chemistry in Relation to Industry," and so forth, for the chemical essay contests that have been conducted throughout the country to stimulate the interest of the high school students in chemistry and science? If you have not read "Chemistry in Medicine," written by the most outstanding authorities in the field, I suggest that every one of you send a dollar to the Chemical Foundation and obtain this India paper, limp, leather-covered edition. It is really a wonderful book. Dr. McCollum and several others have chapters on vitamins and the nutritional situation.

I might add that one of my former colleagues, who accompanied MacMillan on the "Kronkerland" expedition, visited me not long ago. One of the first questions I asked him was in regard to this problem of Eskimo diet, and he told me that he noticed that the first thing the Eskimo ate as soon as the walrus or seal was killed was the warm fresh liver.

We have found, as is known, by experimental work that if there is any one organ in the body that stores all of the vitamin supply of the animal, it is the liver. We find vitamins A, B, C and D loaded up in the liver more than in any single tissue of the animal body, and it was interesting to me, scientifically, to know that that is the first organ practically that the Eskimo wants. The lean meat, the lean muscle free from fat, contains less of all these vitamins, including the fat-soluble vitamins, than does the liver and the other organs, especially the vital organs, of the body.

DR. R. A. BARLOW: After listening to all of these papers (and having worked in the laboratories, myself), I think a note of warning should be given.

One is led to believe from our enthusiasm in reporting all of this research that we have here the solution for the prevention of respiratory diseases, and I, personally, believe that we have struck on a factor. But it is only one factor. I do not think that our message will be complete without a note of warning that just because a child is fed fat, vitamins and the proper diet such as has been outlined here, he is going to be free from respiratory infection. He is not. A certain amount of work can be done to prevent their development, but once a

child has an infection, I do not believe cod liver oil, or butter-fat or anything else is going to stop it. If a sinus infection develops, one has to proceed in a time honored method of attack.

DR. CHARLES W. RICHARDSON: I wish to correct Dr. Barlow. In quoting from my address, he intimated that I thought sugar in itself was the cause of the disease. I stated that sugar, by lessening the appetite for those foods which build up and develop the system, caused the conditions from which persons suffer.

I want to state, from another point of view, that I think an important feature has been brought out in this discussion, and that is how far vitamins influence causation of disease, and how they do it. I think we have proved that the lessening of the amount of various types of food causes diseases and infections (and I mentioned that also in my address) by lowering the resistant powers of the body and the cells of the body, so that the individual becomes susceptible to various types of infections.

I should like to say a word with regard to tonsils and lymphatic tissue of the nasopharynx, pharynx and elsewhere. We were in hopes that something more or less positive would be brought out with regard to the influence of these various disturbances in innervation in producing hypertrophy of lymphatic tissue. I know that I was hopeful of that.

I read in an article recently that by the excessive use of the germinal agents, vitamin A and vitamin B, there was a hypertrophy of lymphatic tissue produced. In connection with Dr. Delavan's paper, I think it is timely.

DR. L. W. DEAN, Iowa City: It seems to me that one thing has come out of the discussion, and that is the extent of our ignorance regarding infections and vitamins, and diets in general. Nutrition does play a most important part in well being, and we must pay attention to it.

Regarding what Dr. Kernan said, I suspect that, just as in the case of the American Indian, these infections were due more to the bringing in of the white man's microbes than to the change in diet.

DR. B. R. SHURLY: It seems to me that Dr. Delavan has given the most timely suggestion, and if we are to summarize and analyze and bring into complete collaboration what we know and add to it, there are three things that are absolutely necessary.

First, we must have a research laboratory with plenty of money back of it.

Second, we must have access to underprivileged children or those who are particularly afflicted with tonsils and adenoids and have a lowered resistance to infection, which we find in the school children of a certain definite age.

Third, we must have a thoroughly scientific clinical collaboration.

DR. D. B. DELAVAN: This is not an idea that can be expected to eliminate for all time diseases of the throat, but if the vast amount of trouble caused by conditions of the throat and these hypertrophic diseases in particular, can be reduced, even to some extent, it will be well worth the effort involved. This will not be accomplished by a committee, a company of men who will study the subject in a desultory way. It will require a comprehensive investigation of the highest possible scientific order.

TONSILLECTOMY INFECTION WITH B. ABORTUS. DR. GEORGE FETTEROLF, Philadelphia.

This contribution is the story of a patient on whom I performed a tonsillectomy, and in whom, four hours after the operation, there occurred a chill and a rise of temperature to 105 F. Physical examination failed to reveal anything abnormal whereby the acute postoperative flare-up could be explained. However, blood studies showed that the boy was suffering from an infection in the blood stream caused by *Bacillus abortus*, a micro-organism known since 1897 (Bang) to be the cause of infectious abortion in cattle. For some years the organism was suspected of occurring in and of being pathogenic to man and in 1924, Keefer, DeKorte and Orpen proved this to be the case. Since then numerous cases have been reported

from widely disseminated sources. The present one is the second to be recorded from Pennsylvania.

This disease has been the subject of a broad study by Kern, and the material used in this paper has been taken largely from his comprehensive study in the *American Journal of the Medical Sciences* for September, 1928,

Human invasion by *B. abortus* is at present, at least among otolaryngologists, so little known as to be a rarity, even a curiosity. This is because what has so well been called the "clinical consciousness" of the medical profession is not aroused to its prevalence. Is it not possible that we all have seen but never have observed cases of this nature? Could it not be that some of those languid, anemic children, with occasional fever, who come to us for examination of the throat and nose, have cases of abortus infection? Is it not conceivable that some of those children who are not cured by tonsillo-adenoid operation by treatment of the sinus and surgical intervention combined with proper hygienic and dietetic measures, have cases of this nature? Children are the milk drinkers of the world, and could it not be that the clinical application of our knowledge of abortus infection might enable one to make a better diagnosis and apply more helpful treatment in the occasional case?

Mercurochrome-220 soluble was given intravenously.

DISCUSSION

DR. C. W. RICHARDSON: The Public Health Service has taken this up vigorously and, as Dr. Fetterolf states, the main idea is to arouse the clinical conscience of the medical profession as to the transmissibility of *Bacillus abortus* through the milk to the human being. Of course, the one great preventive is the pasteurization of all milk.

DR. H. L. SWAIN: I should like to inquire whether the patients in the other cases have been as long in getting rid of that germ as this patient has, and whether anything has been done in the way of finding a vaccine or an antitoxin, by either the veterinarians or the physicians, to combat the disease? If there is an infection like this in the blood, it seems that it can be reached only by something which enters the blood. I do not see how, otherwise, anything that has been done will help the boy get rid of his infection.

DR. F. T. HILL: There were I think, three cases of *B. abortus* fever in the hospital at Waterville, Me., last summer. I became rather interested in them and routinely, began to use the agglutination test for *Bacillus abortus*. It was interesting to find that a larger number of cases showed a positive test than showed any symptoms. It is not a new infection; some of the patients may have had it in the past and still react to tests. There is some question as to the value of the positive agglutination test.

DR. B. R. SHURLY: I should like to ask if mercurochrome was given intravenously.

DR. W. B. CHAMBERLIN: I was interested in the remark of Dr. Hill that the agglutination test is not often of great value, in that so many people react to it. There is one point, however, that I want to emphasize and that is the matter of operating in these cases, in either children or adults, in the presence of a fever. I do not care how many confirmatory recommendations one may have on the part of a medical man, it is always unwise in my opinion to operate on a patient, to remove the tonsils, in the presence of fever.

After all, the tonsil operation, as far as I know, outside of a peritonsillar abscess, is never an emergency measure but always an operation of choice. It can always be deferred and, in my opinion, it always should be deferred until the temperature has been absolutely normal for a considerable period of time.

DR. J. B. GREENE, Asheville, N. C.: Dr. Fetterolf's paper has interested me very much, particularly so since one of our prominent surgeons was stricken with this disease. This physician had under his care a patient suffering from an infection with *Bacillus abortus*. A few months later he developed the same

illness himself. The blood in each case was sent to the Hygienic Laboratory in Washington and the agglutination reaction to *Bacillus abortus* was obtained. The peculiarity in both cases was the irregular type of temperature which continued over a long period of time with frequent intermissions. The characteristic blood count was a low white cell count with relatively few leukocytes, and an increase of lymphocytes. It would be interesting to know if Dr. Fetterolf's case showed this rather characteristic blood picture.

DR. GEORGE FETTEROLF: I was interested in hearing Dr. Richardson speak of the number of articles that have appeared on this subject. As a matter of fact there are many, Kern reviews fifty-seven references in his paper — a classic number.

As regards Dr. Swain's question, I can answer that only in this way. There are thirty-six cases reported in detail. In two of them the patients died; fourteen apparently were cured and the disease is continuing in the other twenty. So, evidently, the disease carried on for some time.

In answer to Dr. Shurly's practical question, I would say that mercurochrome was used intravenously.

Dr. Chamberlin's comment on operating in the presence of fever, I think is entirely correct, and I shall be much more hesitant in the future about doing so. Yet, there are cases of tonsil infection in which the fever is due to the infection and ceases when the tonsils and adenoids are removed. One should consult with the internist, and then use one's best judgment. No hard and fast rule can be laid down.

I cannot answer Dr. Greene's question about the low cell count.

THE USE OF ENDOTHERMY IN LARYNGOLOGY. DR. H. G. TOBEY, Boston.

There are three distinct ways in which the various high-frequency currents delivered by the machine are used in electrosurgery: (1) superficial dehydration or desiccation; (2) cutting, and (3) tissue coagulation.

Anesthesia may be either local or general. When the lesion to be treated is well above the cords, local anesthesia with exposure of the larynx by the laryngoscope is the method of choice. As each application of the current does not exceed a few seconds — five to ten — the procedure is easily borne by the patient. Wherever possible of use, the Mosher laryngoscope is preferable to the Jackson for the reason that the open side allows the electrode to be introduced at an angle and therefore to be more accurately placed.

General anesthesia is preferable for the treatment of lesion of the cords. In conjunction with this, the Lynch suspension is a sine qua non. It requires all the skill which one can muster to apply the electrode to the edge or inferior border of one cord while properly retracting and protecting the other. Mention has already been made of a weak coagulating current in place of the desiccating current in this situation.

The danger of ether explosion seems to be entirely obviated by the complete removal of all ether from the vicinity of the patient and a waiting of at least three minutes before proceding with the application of the current.

The one outstanding advantage of the method is the soft, pliable elastic scar which results.

The indications are malignant disease, angioma, cysts, varix of the vocal cords and papilloma.

DISCUSSION

DR. R. C. LYNCH: I have been working with electric current in the larynx since 1913, using first the fulguration spark, then the actual cautery and finally desiccation and coagulation, and I want to thank Dr. Tobey for bringing the subject before you. I want to supplement what he has said with my own experiences.

I have found, for instance, that in work between the vocal cords the ordinary wooden spatula which is used for a tongue depressor is the best implement for

spreading the cords apart. I have a long handle into which this tongue depressor fits, such as the handle that comes in the box in which they are sold, and I have made that long enough so that with the addition of the wooden spatula I trim it down with a knife, varying in width, depending on the width between the vocal cords that I want to spread. I put it in vertically and turn it flat. This implement is much better than the long bivalve speculum which I showed to this society in 1915.

At the present time I have four cases of intrinsic carcinoma involving one vocal cord in which the tumor, according to my conception, has broken through the basilar membrane but has not progressed to the degree of interfering actually or totally with the function of the cord.

I have four cases that come under the classification of tumors in which I have destroyed the growths by coagulation. In the last, nearly two years have elapsed without recurrence. And there are two cases of carcinoma of the base of the tongue in which there is no apparent palpable glandular enlargement in the neck and apparently here, too, the tumors had not invaded so deeply into the tissue of the tongue. The patients in these two cases are also well without recurrence up to this time. In the last one eight months have elapsed.

This is the first time that I have spoken of any of these cases because I was still undecided whether to put these cases on record because I was not sure, whether the condition would recur shortly after I had reported the cases.

Relative to the papilloma, I am sorry to say that my experiences cannot approach those of Dr. Tobey. I have used desiccation and coagulation after the method that he has described.

DR. H. B. ORTON, Newark, N. J.: I have been very much interested in what Dr. Tobey has stated and in Dr. Lynch's experiences.

I have been using diathermy for malignant diseases of the larynx and upper air passages in the last few years, and I wish to emphasize the danger of using a general anesthesia in these cases in which surgical diathermy is being used.

Notwithstanding the fact that all precautions were taken, I have had two explosions with ether, fortunately without bad results. I would recommend that rectal anesthesia be used, as I have had no such bad results when this procedure was followed. The two explosions that I had have been with ether when administered through a tracheotomy tube.

DR. D. B. DELAVAN: Surgery is indeed often slow in adopting new ideas, as in this matter. Endothermy was discovered in New York by Dr. Finley R. Cook in 1903. He evolved the principle, invented instruments for its application, tested them successfully before witnesses on numerous varied types of cases and abundantly proved the value of the new method. In the *New York Medical Record* for Dec. 21, 1907, he published a full account of his work. This much credit should be given him for what, after twenty-five years, is becoming so popular.

DR. M. C. MYERSON: As one who has had a limited amount of experience, but yet a fair experience, I might say there are certain phases of the use of this current that one must be acquainted with and be careful about. For instance, I do not believe Dr. Tobey mentioned in his paper that when the current is on, any part of the patient's body that the electrode touches becomes desiccated or coagulated or eburnated or carbonized.

For that reason, electrosurgery, unless electrodes of unusual precision can be developed, has a limited field; in fact, it probably has no field at all in intranasal work.

In my own experience, I have never attempted to use an electrode within the nose. When there is indication for electrosurgery in a condition affecting the nasal passages, that condition should, from the standpoint of safeguarding the healthy tissues, be attacked by an external incision and a wide exposure.

One must not forget that in soft tissue that is more or less vascular, there are many blood vessels that are uncovered when the slough comes away. For that reason it is questionable whether diathermy will ever be accepted universally in tonsillectomy.

I can readily appreciate that an extensive lesion within the larynx or about the larynx can be safely attacked with a diathermic electrode, but I cannot subscribe to the use of an electrode of any type for a simple hemangioma or a simple neoplasm that is on the surface of the cord and that is small.

Compare the use of diathermy with the use of a suitable forceps for the removal of a small neoplasm. It does not matter whether the forceps touch any structure within the larynx, nor does it matter if the forceps grasp a part of the cord that does not contain the neoplasm. One can always unloosen one's grasp and take another. But if the current is on, there is a destructive effect. In using a long flexible instrument in the larynx, one will have to see the electrode to apply it to the neoplasm. This would make the electrosurgical technic a dangerous thing, except in fairly extensive lesions within the larynx.

I have had occasion to desiccate a carcinoma of the trachea by this method, where the carcinoma was secondary by extension from a thyroid involvement, with fairly good effect.

I favor conservatism and caution in the adoption of this technic for every condition in rhinolaryngology.

Dr. H. G. Tobey: As to papilloma of the cord, I should defer to Dr. Lynch's experience, which is vastly greater than mine, but it has been my observation so far that the recurrences have not occurred when I had a good desiccation and a good scar.

I was glad to hear Dr. Delavan's remarks concerning Dr. Cooke.

Dr. Myerson got away from the subject a bit. Of course, we cannot discuss carcinoma of the tongue and tonsillectomy at this time. I do not believe in diathermy for tonsillectomy, but the advantages of diathermy in carcinoma of the tongue, in safety of removal and possibilities of recurrence, I think vastly outweigh the possibilities of secondary hemorrhage.

Of course, it is not to be understood that I am advocating this for universal application but only for certain selected cases.

Report of Several Cases. Dr. R. McKinney, Memphis, Tenn.

The pathologic lesions (with lantern slides) in two cases with autopsy reports were illustrated. The first was a case of a cockle-burr in the left main stem of the bronchus. The bronchus was completely obstructed. The child had bronchopneumonia and also an abscess, but the cockle-burr was the cause of death; it should have been removed. I was out of town when the case came to my service, and my assistant was afraid to give ether anesthesia as the boy was in a serious condition, but I think that if the cockle-burr had been removed, the boy would have recovered. I have never before seen a cockle-burr in the lungs. It usually stops at the vocal cords. Most of you have had cases like this, especially those who live in the sections of the country where cockle-burrs are seen. I have had at least a dozen cases of cockle-burrs in the larynx, but this went down into the bronchus, and trapped the air in the lung. The patient had more or less of a pneumonia with it. Death occurred twelve days after he had been admitted to the hospital.

The second case was in a woman about 45 years of age, who had a definite history of cardiospasm. Esophagoscopy was done and a stricture that could not be passed was found, but she had developed pneumonia while in the hospital, and died.

On autopsy, the following condition was found. There was complete stricture of the esophagus, even after death.

We regard a cardiospasm as a nervous reflex — it is so regarded by a great many — but in this case the spasm was continued after death. We naturally thought it would relax, but it did not, and we got through it with a great deal of difficulty.

DISCUSSION

DR. W. B. CHAMBERLIN: It has seemed to me for a long time that in these cases greater co-operation is needed between the dentist and the rhinologist. I know at home, at least, that there is a decided tendency on the part of the dentist to disregard the help which the rhinologist can give him. I am not at all sure but that the same attitude prevails on the part of the rhinologist.

In support of the statement which I have just made, I wish to cite two rather typical cases, one of which was a reflection on the dentist and the second was, as will be seen, rather a reflection on me.

The first case was in a woman who had had an antral infection, evidently from a diseased tooth. The dentist, without making a counteropening into the nose, had treated this antrum through a small fistula in the alveolar process, irrigating some three or four times a week for a period of eight months. On irrigating through this sinus, when the patient first appeared at my office, I found the antrum filled with a foul-smelling pus, and concluded that the only thing which would give her relief would be a Caldwell-Luc operation. This I performed under local anesthesia and within a few weeks the antrum was clean. The fistula in the alveolar process still remains.

The second case was that of a boy, aged 18, the son of a former colleague. He came in with the left side of his nose filled with pus, also bringing a roentgenogram with him, which showed a cloudy left antrum. Irrigation, which was rather difficult on account of the character of the pus, showed a foul-smelling pus with a predominance of a caseous material, almost the consistency of a thick Dutch cheese. I irrigated his antrum two or three times and determined on a window resection of the naso-antral wall. He failed to appear at the appointed time, on account of illness, and did not reappear until several days later for this operation.

When he came in, he showed me the one entire root of a molar tooth and wondered if that could have any bearing on his condition. He also said that he had blown practically no pus out of his nose since he had expelled the root of this tooth.

I looked in his nose and found it practically normal, with no evidence of pus. In spite of this, I determined to make another exploratory puncture, simply to make assurance doubly sure. I did so, washed out the antrum, and found it perfectly clean. This was one of the foulest antrums that I have ever encountered. I washed out not the typical pus which one usually gets, but almost solid masses of dense, caseous material. And yet, in spite of the foul character of this discharge, the disease promptly cleared up, after the root of this molar tooth had been expelled.

DR. R. H. SKILLERN, Philadelphia: I cannot get the information from transillumination that the author seems to acquire, and I do not believe that my experience warrants giving such a high percentage to dental infections as Dr. Berry has.

This has been a moot question, of course, for a good many years. I do believe that in these large antrums, particularly those in which the condition is chronic and in which it has been present for any length of time, the root of the tooth can be infected from the antrum and that one can have a secondary dental infection from the primary infection of the antrum which was of a nasal origin. Of course, this naturally excludes all teeth that are dead or those that have demonstrable abscesses of the roots. I think all of us now look on such eventualities as foci of infection, and they should be done away with regardless of anything else.

It seems to me, at least, from my own experience, that these cases of antral infection from dental origin can be placed closer to, and perhaps under, 10 per cent than over that figure. I feel that there have been a great many healthy teeth needlessly sacrificed in an endeavor to cure these old chronic antral conditions.

NONTUBERCULAR LESIONS OF THE LARYNX IN THE TUBERCULOUS. DR. J. B. GREENE, Asheville, N. C.

On account of the great frequency of laryngeal tuberculosis in tuberculous persons the condition should be regarded as tuberculous until proved otherwise.

However, patients suffering from pulmonary tuberculosis have no immunity from other laryngeal conditions, particularly simple laryngitis, syphilis and cancer. A careful history of such cases should be taken, and a close study should be made of the larynx over a period of time before making a positive diagnosis other than tuberculosis of the larynx. The situation of the lesion is important in making the diagnosis, particularly so when laryngeal tuberculosis and cancer are being considered. Laryngeal tuberculosis is prone to attack the posterior part of the larynx, particularly the commissure and the end of the cords, while cancer is most frequently found near the middle or anterior part of the cords. In making the diagnosis of syphilis of the larynx in tuberculous persons, one must bear in mind the infrequency of its occurrence. The Wassermann test, while not diagnostic, is helpful in such cases.

The question of biopsy in tuberculous persons is rarely indicated and seldom justifiable. A careful history of such cases with a study of the appearance of the larynx over a period of time will usually make the diagnosis clear. Laryngeal tuberculosis may run a favorable course after biopsy.

DISCUSSION

Dr. T. J. Harris, New York: I feel that the present generation of laryngologists have been and are remiss in the matter of attention paid to laryngeal disease. Those who preceded us spent a great deal more time on this. They studied their cases much more carefully than we do. They did not have the direct method, but they used, with a great deal of skill, the indirect method, and I am of the opinion that as a general proposition they knew more about the larynx than the majority of laryngologists know today.

When the writer makes a statement as to the large number of cases of involvement of the larynx in tuberculous diseases generally, I listen with a great deal of interest. I had not thought that the percentage was so high as he put it, but I am wondering whether one can speak with an extreme degree of certainty as to just how many cases there are and just how far one can make a lasting and definite diagnosis.

Dr. Greene gave an illustration, at the close of his paper, of mixed infection. Some one, one of our English confreres possibly, has made the statement that he did not believe it was possible to make a positive diagnosis of laryngeal disease between cancer and tuberculosis without biopsy.

In a recent meeting of the New York Laryngological Society, considerable time was spent discussing one or two laryngeal cases that were presented. Perhaps Dr. Swain will, or has already referred to a case which he presented in which there seemed to be up to the last and even after the autopsy, a question about the diagnosis.

Dr. G. B. Wood, Philadelphia: As a rule the early symptoms of laryngeal tuberculosis are the result of functional disturbances, that is, interference with voice production. Pain is a prominent symptom only when the lesion is extrinsic, involving the epiglottis or arytenoids. It must be remembered, however, that aphonia frequently occurs in cases of pulmonary tuberculosis which are of a functional nature and not due to presence of local tuberculous disease.

It is essential that every patient with pulmonary tuberculosis should have repeated examinations of the larynx as the detection of early lesions is of great importance as to the prognosis. Simple laryngitis, either acute or more or less chronic, is frequently seen in pulmonary tuberculosis and this cannot be wondered at when one considers the amount of infected sputum that is being continually coughed up out of the lungs onto the surface of the laryngeal mucosa.

The importance of these pyogenic infections of the larynx in the etiology of laryngeal tuberculosis has been pointed out some time ago by Blake of London. He showed that there frequently occur minute intra-epithelial abscesses, the result of pyogenic infection, and that these abscesses are probably one of the important

etiologic factors in the inoculation of the laryngeal mucosa with the tubercle bacilli.

DR. J. A. BABBITT, Philadelphia: There is one thing that I should like to ask on the question of, not necessarily complete paralysis, either unilateral or bilateral, but the mobility in the cords. I should like to have Dr. Greene tell me whether he has made any observation which has been helpful on these two or three occasions, as to the mobility of the cord.

DR. B. R. SHURLY: I feel that this paper of Dr. Greene's is along the line of stimulating just what Dr. Harris has mentioned, and that is the fact that there seems to be an influence among the younger men to ignore their scientific interest in diseases of the larynx.

Following statistical reports for many years, I find that in the institutions at Detroit the percentage of laryngeal tuberculosis in the sanatoriums is almost constantly, from year to year, around 15 per cent. It runs 17, 16, 14 and 15 per cent.

I have yet to see, in all these years of experience, a single case of laryngeal tuberculosis that did not have some pulmonary focus or some tuberculous focus elsewhere. It is therefore entirely unnecessary to do a biopsy.

DR. C. W. RICHARDSON: We must stimulate the younger generation to look at the larynx, to study it, and to learn that differentiation and diagnosis and treatment are possible in conditions of the larynx as well as other parts of the upper air tract.

DR. J. B. GREENE: Dr. Harris spoke in reference to the frequency of the occurrence of the mixed infections, tuberculosis and syphilis. In my experience these two infections occur together infrequently in the larynx.

Dr. Wood mentioned the interesting occurrence of hysterical aphonia in the tuberculous patient. This does occur in a fair degree of frequency and should have been mentioned in the paper. This occurrence of hysterical aphonia in the tuberculous patient is especially likely to happen in sanatoriums where association is rather intimate with patients suffering from tuberculous laryngitis.

Dr. Babbitt spoke of the defective mobility of the cord in cases of laryngeal tuberculosis. It is difficult to say whether the paralysis is due to the presence of tracheobronchial glands or pleuritic adhesions.

Dr. Shurley spoke of primary laryngeal tuberculosis which we have come to accept as nonexistent. Laryngeal tuberculosis is a complication of pulmonary tuberculosis, though at times a careful examination of the chest is required to detect it. At times, it requires stereoscopic roentgenograms interpreted by one of considerable experience.

MIXED TUMORS OCCURRING IN THE SOFT PALATE, WITH REPORTS OF TWO CASES AND A SURVEY OF THE RECENT LITERATURE. DR. ROBERT SONNENSCHEIN, Chicago.

The so-called mixed tumors which occur frequently in the cephalic region have given rise to a good deal of controversy because of the uncertainty regarding their origin. These tumors are characterized by the heterogeneity of the tissues contained in them as well as by a great irregularity in the arrangement of the cells.

The most important clinical fact connected with these tumors is their long duration and slow development. As a general rule, they exist for many years before the patient consults a physician regarding them. Tumors in the soft palate are comparatively rare. Altogether only about fifty cases have been reported in the literature. In the parotid and submaxillary regions they are found much more frequently.

According to McFarland, the following tissues may be found in mixed tumors of the salivary glands and of the neck:

A. Connective Tissues:

1. Cartilage of varying types
2. Mucous tissues of various types
3. Fibrillar connective tissue
4. Bone (rare)
5. Adipose tissue (very rare)
6. Muscle tissue (very rare)
7. Lymphoid tissue (very rare)

B. Epithelial Tissue:

1. Glandular epithelium, almost always
2. Squamous epithelium, in the majority of cases

Origin and Structure of Mixed Tumors

1. The endothelial theory (Volkmann) which held that the tumors were derived from the endothelium of the lymph vessels.

2. The embryonic theory which is that the tumors are derived from pluripotential cells which have ceased to develop further at different stages of embryonic development.

3. The branchial theory which was developed largely to explain the presence of cartilage.

4. The theory of origin from embryonic gland germs.

5. The theory of origin from fully formed gland tissue. According to this theory (which Fry states is becoming more and more adopted) these tumors are derived from adult gland cells.

DISCUSSION

DR. R. C. LYNCH: I want to put on record a case of what seems to me to be a mixed tumor of the parotid type, if Dr. Sonnenschein will accept that, growing from the posterior aspect of the larynx, subglottically and extending down the trachea, extending so far down the trachea that in order to remove it completely I am afraid it will be necessary to enter the mediastinum.

The case was referred to me by Dr. Shea. I first saw the patient in 1924, and as I said before, we did a bronchoscopy or tracheoscopy in order to see the tumor, but when we found we could not get at it that way, a thyrotracheotomy was performed, and 50 mg. of radium used for eight hours. To our surprise, the tumor melted away.

The patient came with a fixation of one cord, lagging of the other cord and interference with respiration. The tumor disappeared for about eight months, when he returned again with an apparent slight involvement. At that time we put on 200 mg. of radium externally, and had to do a tracheotomy. We used the rubber tube so as to get away from the secondary irradiation of the radium, which is a precaution that should be followed.

The tumor melted again. Then we saw the patient in February, 1928, and at that time he had a third recurrence, the tumor growing from around the tracheotomy tube. I cut off this section, and it looked like cutting a piece of Irish potato. It did not bleed at all. We put the radium on again, putting 50 mg. into the tumor and 150 mg. outside, cross-fired, and to our great surprise, the patient was again breathing through his larynx by the natural passage and had the use of his vocal cords when we saw him on April 19, 1929.

DR. W. B. CHAMBERLIN: Dr. Sonnenschein has kindly referred to a case report which I made before the Triological Society in Atlantic City, I think about two years ago. This was in a woman, aged 40, on whom I had previously performed a tonsillectomy. The patient's husband, a physician, brought the case back to me because a local physician in Virginia had diagnosed the condition as peritonsillar abscess.

At the time of my examination, I could find no remains of tonsillar tissue. This tumor was discovered entirely by accident, in trying to remove what I thought was a small enclosure of tonsillar tissue in the cicatrix. This tumor was large, measuring 5 or 6 by 7 cm., and extended up to the base of the skull. The lower part of it was discrete, but the upper part was divided into two lobes which were separated by the styloid process.

I removed practically the entire tumor easily. It was not a difficult operation because it shelled out almost entirely in one piece. The other piece on the inner side of the styloid process, consisting of probably one fourth or one fifth of the entire mass, was shelled out easily also and was removed with the snare.

When I had removed this tumor, I could see the styloid process and back of it the facial nerve apparently dividing the upper part of the fossa into two fairly equal divisions. I had never seen a tumor of this type before and did not know what I had until the histologic examination was submitted to me.

Strangely enough, I saw two of these tumors inside of two days, shortly before coming away. All of these tumors, I might say, were on the left side. I think Dr. Sonnenschein's were on the right.

Clinically and operatively, these tumors are not malignant, but purely benign. They are surrounded by a dense fibrous capsule. They shell out readily and show no evidence of metastasis. In none of these cases was there any evidence of involvement of the cervical lymph nodes.

Dr. J. F. Barnhill: I wish to speak only as to the malignancy and non-malignancy of these tumors. I have not seen a tumor of the soft palate of the character described. I have seen and operated on seven of the parotid gland, some of which were extremely large. Some of these were operated on as long ago as fifteen years, and there has been as yet no indication of recurrence.

I am asked if I ever had facial paralysis occur in these cases. That is a good question. I did not. One can promise such patients, even though the mixed tumor of the parotid is large, that they will not have a facial palsy. This, of course, is provided that the capsule is complete and that during operation the surgeon keeps close to the capsule, because as the tumor enlarges, the capsule presses all branches of the nerve aside, and so far as careful observation could determine, no nerve of any considerable size ever enters the capsule of the mixed tumor.

Dr. R. Sonnenschein: With reference to Dr. Lynch's case, histologically it was a mixed tumor.

So far as I can recall at present, in going over this literature carefully, I do not remember a description of any case in which the condition was located just where this was.

As Dr. Barnhill has said, as long as the capsule is preserved intact there is practically no danger of recurrence. But on the other hand, it should also be remembered (because that view is apparently shared by the best observers quoted in the paper) that although the histology leads one to believe that it is a malignant tumor (these two that we had were mixed carcinoma, as judged by the microscope), it is the clinical history that counts. While it does happen that these tumors recur or even cause disturbances in their locality, they do not produce metastasis. It will be recalled, no doubt, that the giant cell sarcoma of the bone which arises from the medulla of the bone is not malignant—histologically, yes, but clinically, no.

Calcification of the Meninges Coincident with Nasal Pathologic Changes. Dr. James A. Babbitt, Philadelphia.

This paper literally refers to coincidence, not etiology. A review of the literature indicates great interest in the past 150 years, though concensus doubts any serious significance. The calcification problem embodies much physiochemistry, though there has been comparatively little research. This paper presents a case

in which intranasal surgical intervention seems to have alleviated a severe and interesting type of atypical neuralgia. The conclusions show the failure in establishing definite correlation.

DISCUSSION

Dr. J. F. Barnhill: It is probable that the cause of the headache or neuralgia in Dr. Babbitt's case lay in the nose or adjacent sinuses. The neuralgia was undoubtedly of minor type. Patients with neuralgias of this character are almost invariably relieved or cured by operation for removal whether the process is located in the teeth, nose, throat or ear.

In discussing neuralgia of this type, and especially in discussions of treatment for headache and neuralgia, another variety of the disease should be mentioned, and some essential points connected with its pathologic process and treatment emphasized. I refer to major neuralgia or tic douloureux. In the type of neuralgic pain referred to by Dr. Babbitt, pathologic changes can be found which account for the pain, and the removal of the pathogenic areas is curative. Such, unfortunately, is not true of tic douloureux, for no pathologic change can usually be found, and no operation on the sinuses, teeth or nose has proved beneficial. Despite this well proved fact, most persons suffering from tic douloureux continue to be operated on.

It has been my privilege to see several hundred cases of major neuralgia, to operate in many, and to make injections into the gasserian ganglion in many. In almost every case the sinuses had been operated on, the septum straightened, the tonsils removed or the teeth all extracted, all to no purpose, for the reason that the pathologic change lies in the gasserian ganglion and not elsewhere. In such cases the posterior root of the ganglion should be severed, or alcohol should be injected into the ganglion itself, for these measures only will cure the tic.

Dr. G. M. Coates, Philadelphia: A year or so ago, Dr. Failer warned us specifically against operations in these cases of calcification of meninges coincident with nasal pathologic change. His reason for doing so was that in one case in which there was some evident calcification, and in which an ethmoid operation was done, the patient died subsequently with meningitis. It has always seemed to me that his point was not well taken in these cases, and that whatever the etiology of the calcification might be, it could hardly be construed, in the majority of cases, at least, as a positive contraindication for operation.

As Dr. Barnhill says, the neuralgic pain in this case, I should suppose was not due to the calcification but possibly to some sphenopalatine irritation from the evident postethmoiditis that was present.

Anaphylaxis and Allergy in Rhinology. Dr. Francis P. Emerson, Boston.

Biologically, it has been shown that after the amino-acids have been elaborated by the action of the enzyme on the foreign protein, any remaining protein not so acted on is exceedingly poisonous. The fact that antibodies have not been found in the allergies is a biologic problem, as the symptomatology is the same as in anaphylaxis. Most of the literature concerns the nonbacterial allergies, with attempt to establish immunity or to remove the patient from the presence of the exciting antigen.

In the bacterial allergies any operation is only a beginning of any treatment. There is always present an endocrine sympathetic syndrome. There is also the question of reinfection, bad mental and physical habits and usually bad family environment that must be taken into consideration.

Dr. H. G. Tobey: I think perhaps we can say once an anaphylactic or allergic or asthmatic person, always one.

We do not know what asthma is. We do not know what anaphylaxis is. It seems to be a congenital condition. One focus of infection in the antrum or the tonsils may be the trigger which starts the asthma going. Clearing that out may stop the asthma. We do not know what next will bring it up.

DR. L. A. COFFIN, New York: We do not know what anaphylaxis and allergy are in a way to make concise and precise definition, but we know it for practical purposes.

I have one patient who has been under my special care for at least twenty years. He has had asthma for the greater part of that time. His particular symptoms were his asthma, from which he suffered daily, and a tachycardia. He had a pulse that counted 150 most of the time. Regulation of diet, high colonic irrigations and acidophilus milk improved this patient so that his asthmatic attacks were infrequent. The tachycardia has disappeared, and his pulse is now 74. Internists who have seen him in consultation have not recognized the anaphylactic or allergic condition.

DR. C. W. RICHARDSON: The great trouble with the allergists, like all advocates of new divisions of medicine, is that they claim their field, their pathology and their treatment are the only true gospel to be employed in these cases. Allergy is an inviting subject. Some of their investigations seem to hold for a time.

The question of heredity is an interesting feature in all cases that we are speaking of. It is not the transmission of a diseased condition from parent to child, but it is implanting on the cells of certain mucous membrane, tissues and so forth, a susceptibility or an inherent weakness to certain impulses which are transmitted into disease pathology. And that is all we can say about heredity.

A child of tuberculous parents has no more change, absolute change, in the protoplasm of his cells than a child born of healthy parents, but there is something indefinable that is implanted which makes those cells susceptible to changes. The same thing is true, I believe, in connection with these disturbances. Is this heredity enhanced by antigens and where are they produced? There is the crux of the whole issue: to find out wherein that impression is made and to get rid of it. It may be, as Dr. Emerson has stated, in the sinuses. It may be in the intestinal tract, by fermentation, intoxication, parasitism, and so forth. Or it may be and probably is, largely, from the proteins which are taken into the alimentary canal and which are not digested.

All of these factors are a great hodge-podge which we have to eliminate and study. I do not believe in one theory with regard to this condition. One factor is eliminated and the patients improve wonderfully. Another one comes in and the patient goes to pieces again. And that is the history of these cases. Some one has said "once a sinusitis, always a sinusitis, possibly with reinfection." So it is in these cases. Once subject to allergy, probably always an allergist.

The fundamental question is what produces in certain apparently normal persons, a susceptibility to certain protein while in other normal subjects there is a perfect immunization to protein infection.

DR. G. M. COATES: During the last two years my associate in the Graduate Hospital at Philadelphia, Dr. Matthew S. Ersner, and I have been investigating the maxillary sinuses, particularly in a number of cases of asthmatic persons, studying them by ordinary methods and also particularly by injections of lipiodine into the maxillary sinuses, and making an x-ray picture not only to determine the opacity and thinness of the mucous membrane, but also to determine the time it took to eliminate the opaque oil.

We have found in our asthmatic subjects, in a great many of them, at least, this same condition that Dr. Emerson has described. There is often a fibrosis of the membrane of the maxillary sinus, and these cases, after radical removal, have apparently done well.

We have also discovered something else that Dr. John Culmer, our pathologist, tells me has not been reported on, though we have not had time so far to look up the literature, and that is the presence of eosinophilin in the diseased mucous membrane of the maxillary sinuses of these asthmatic patients. Of course, we all know that the eosinophilia is a common observation in asthmatic persons. What its relation to asthma is, I do not know, and I do not know that any of us

know exactly. And particularly, we do not know what the presence of the eosinophilia in quantity in the mucous membrane of the maxillary sinus of asthmatic persons may show or whether it shows anything, but I should like to ask Dr. Emerson whether he has noted that at all, or whether he knows of its having been noted before. We contend that it seems worthwhile to present the observations in more detail later.

Dr. H. L. Swain: Early in my career, I once was guilty of publishing a paper the title of which was "Asthma, Its Intranasal Origin and Surgical Treatment," which was a bold assertion, but it seemed unquestionably to be proper. That led me to study the pathologic changes in the region, apropos of something existing back of the whole thing, and I am sure that people with hyperesthetic rhinitis or people who can develop polyps and hyperesthetic rhinitis without any pus and who can develop a dry hypertrophy of the membrane within the sinuses, show tissues under the microscope histologically different from those who never develop a polyp or hyperesthetic rhinitis.

So much was I convinced of this that at one time I made the statement that I could tell by a microscopic slide whether the tissue came from a person with hyperesthetic rhinitis or a person with a tendency to anaphylaxis and allergy, as we now like to call it; that I could tell by the tissue, itself, as compared with hypertrophies of the type taken from other noses not so inclined.

Now then, if we have gone from the purely physical into the realm of, we might say, the hypothetical in dealing with anaphylaxis and allergy and inheritance, which Dr. Richardson was talking about, those notions but emphasize the fact there must be an absolute difference in the make-up and organization of the two different types of individual.

Dr. F. P. Emerson: I was much pleased with what Dr. Richardson had to say about the basic conditions in general medicine and hereditary influences, which can be nothing but predisposing.

The influence that led me to become interested in this study was the fact that when a patient presented himself as an asthmatic subject, I took the history starting from childhood. I asked, "How many years were you well?" and "What did you have then that seemed to be the starting point of your present condition?" I had them tell their own history in a general way up to the time of visiting me. As a result of following this procedure I was impressed not with the conditions that led me to think they had inherited any predisposition to unstable cell conditions, but with the fact that at some definite time, either when they were 1 or 2 years old, or subsequently, they had an infection or condition like the measles or pneumonia, or grip, and following that they had asthma, never having had it before.

In considering this hypersensitiveness, we all admit that the exciting causes that may precipitate any one attack at any time are innumerable, but the structural basis of the cell condition that makes that hypersensitiveness is a broad subject, and this is offered only with the idea that it may excite somebody to contribute something further, because we are not getting anywhere in regard to our understanding of bacterial allergy.

In the nonbacterial types, whether the patients have an inherited predisposition or not, they succeed in establishing immunity, and in the bacterial types, if one finds and corrects the pathologic process, the patient's resistance will at least be increased. Life will be made more livable, and incidentally, if the asthma is cured, a great deal has been accomplished.

I think that "cure" is an indefinite term, and I think, as I said in my paper, that if it is due to a bacterial hypersensitiveness, whenever the patient has an acute infection it is going to start up. It may be one or three years from the time of operation, but if they have an attack only once a year instead of once or twice a week, that is a tremendous gain.

TREATMENT OF MAXILLARY SINUSITIS. DR. JAMES J. KING, New York.

The subject is divided into the treatment of acute, subacute and chronic maxillary sinusitis, and some mention of malignant disease of the maxillary sinus. Daily irrigations have been successful in clearing up most acute conditions of this sinus. In the subacute and chronic cases, the window operation in the nose, giving drainage and ventilation to the maxillary sinus and insufflation of iodine powder, has been helpful in the treatment of these conditions.

DISCUSSION

DR. B. R. SHURLY: This is one of the most interesting and scientific problems that the rhinologist has at the present time. Not only are the scientists infected with the idea, but the laity are also infected with the idea of going to a specialist in diseases of the sinus.

Of course, we at once divide cases of maxillary sinusitis into two distinct classifications: the acute and the chronic, and what is done in an acute case is entirely different from what is done in a chronic case. A series of washings or one only will frequently be sufficient for an acute case. An enormous number of antiseptics are used. We change off from one to another, and there is a great question as to which is better, the salt solution or mercurochrome.

It seems to me that we often overlook the wonderful value of ephedrine in 3 per cent solution. This is a decided modern advance in the shrinking of the turbinate bodies. It does away with the irritation following the use of epinephrine, and there is soon to be put on the market a synthetic preparation of ephedrine, which I have tested out for one of the great laboratories in Detroit. By using this preparation we can have the turgescence removed for a period of from four to six hours, which is different from the short duration of epinephrine, which is a few minutes, fifteen minutes or so. The synthetic preparation will reduce the price of this product decidedly. It seems to have the same efficiency in shrinking the membrane and the turbinates and seems to have the duration, but it is apparently worthless in operative procedure because it does not control the hemorrhage in the same way that epinephrine does. It will soon be on the market as a much cheaper preparation to use. I believe that the use of ephedrine in acute cases is a valuable therapeutic advance.

It seems to me that the pathologic change in chronic cases is the criterion on which one must use one's surgical judgment in the selection of procedure, and while the window resection is of extreme value and undoubtedly should be the first choice in the great majority of cases, and will be sufficient in more than 70 per cent of the ordinary infections that are seen that are beyond the acute stage, the subacute, or into the chronic stage; of course, when there is a more extensive pathologic process, with polypi formation and the chronic, infected antrum, one must go into the problem much more deeply and much more carefully than simply with the window resection. The question of infection from the teeth has been carefully gone into and must be given careful consideration.

Our knowledge of these conditions has been greatly advanced by roentgenology. It seems to me that there is great difference in the scientific deductions from and reading of these plates. There are an enormous number of poor x-ray films put out in our field. The ordinary roentgenologist is not always efficient in reading these plates, and I look for from 10 to 20 per cent error in the reading of the plates. I am much more satisfied with clinical differential diagnosis and am only satisfied with that in confirming the x-ray observations. I believe we have given too much over to the roentgenologist, and that we have less today of clinical intuition and clinical knowledge which the older laryngologists had to depend on absolutely, for they had nothing else.

DR. L. A. COFFIN: I congratulate Dr. King on knowing when to stop operating. When symptoms are relieved and nothing remains in evidence of disease but an x-ray shadow, it is well to allow the patient to remain at home.

It is not an infrequent procedure of my own after the window resection, to teach a patient how to irrigate his own antrums. It is sometimes necessary to do a radical operation. I do not recall a case in which the patient has failed to recover after this procedure.

DR. D. MACPHERSON, New York: I should like to stress the point (there are a great many points in Dr. King's paper that one could comment on) about the preoperative stage in acute cases, the stage in which it is not necessary to do anything, at least, not to irrigate, for irrigation may be harmful in the first four or five days. I think that many patients treated without irrigation, given ephredine, as Dr. Shurly has suggested, and given the attention that should be given a common cold, make a better recovery than those in whom irrigation is done. I think irrigation does harm in such cases.

DR. F. P. EMERSON: In acute conditions, as the last speaker said and as Dr. Shurly said, I do not think that we get an acute condition in the antrum without changes in the nasal mucosa in which the ostium becomes blocked. If we use the mild silver protein packs and the ephedrine and direct our measures to the nasal mucosa and to getting the ostium opened, it is sometimes not necessary to touch the antrum, and it is safe to do that for the first forty-eight hours, at least. And then if the antrum must be opened, I cannot see the surgical indications for a small needle puncture. We have, really, an enclosed abscess with the ostium blocked off, with the pathologic change confined largely to the sinus mucosa which is enormously swollen. I immediately make an opening large enough beneath the inferior turbinate and see that the opening through the mucosa is also adequate, and then leave it alone, because, as the last speaker said, every time one irrigates a nasal mucous membrane that is edematous, one simply irritates it and keeps up a continual discharge, whereas if the opening is sufficient and adequate and there is ventilation, the result will be much better. Treat the abscess the same as an abscess in any other part of the body and continue to treat the nasal mucosa and get the lymphatic and circulatory processes relieved, which indirectly relieves the maxillary mucosa.

When it comes to the chronic cases of maxillary sinusitis, it is a question, after the pathologic process has left the sinus mucosa and involved the connective tissue, for surgical judgment as to whether the fibrosis has gone beyond the stage of recovery or whether palliative measures can be used, with the idea that that antrum may clear up.

I think right here is the point, that, as some one remarked in the discussion of my paper, once a sinusitis, always a sinusitis, because when that fibrosis has gone beyond a certain point, it never clears up. The symptoms are relieved and the patient is better, until his resistance is lowered by some condition later, and then that is a potential trouble for general systemic conditions.

If the sinus infection is not of long duration, I seldom use a window resection, but I continue treating the nasal mucosa. That improves the circulation, and I think does a great deal toward improving the general condition. When I think the fibrosis has become diffused, as it has in all sections that I have made, and never circumscribed, I do a radical operation to clear up the entire chronic infection, and since we know that the sinus membrane lining regenerates, we replace what has been the chronically infected membrane with a new membrane that extends in from the nose.

DR. G. E. SHAMBAUGH: I cannot help but ask myself the question, "Why do we attach so much significance to pathologic changes in the antrum when the same thing takes place in the mucosa of the nose?" We meet plenty of cases of hypertrophic rhinitis and atrophic rhinitis. Do we think we have to ream out the membranes of the nose because there is a pathologic membrane there? Why attach more importance to a pathologic membrane because it happens to be in the antrum?

DR. J. F. BARNHILL: I wish to say that I thoroughly agree with those who do not practice prolonged irrigation in the treatment for acute cases.

Dr. King said, as I understood, that thirty days was his average time in obtaining relief or cure. In thinking over my own experience, I am pretty sure that in cases in which treatment consisted of keeping the nose open by means of ephedrine and but little irrigation of the sinus, the patients were well much sooner than thirty days.

I wish also to say that I thoroughly agree with those who, when they find sufficient pathologic change in the sinus, open the sinus, actually see what is within, and then remove whatever pathologic process they see. As time goes on I believe more and more in the efficacy of radical operation in chronic cases in which there is sufficient pathologic change present to require an operation, and consequently less and less in the window resection as the best means of surgical treatment.

One thing that has not been mentioned and that ought really to be is this: we know that general infection, secondary general disease, arises chiefly from just such foci as are now being discussed. Leading internists have said over and over again that heart conditions of an inflammatory type, rheumatic infections, kidney ailments and in fact a surprisingly large number of general diseases which give the internist most concern, arise from primary foci in the head. Should we overlook this fact, and should we manage these foci by incomplete methods of eradication, we should lose an opportunity to prevent secondary septemic involvement of a dangerous nature, and should hereby miss an opportunity for doing good which will not again be given. Therefore, drainage and removal of a pathologic process at as early a time as can be done is really what the internist of today expects of us, and what he sometimes criticises us for not doing.

Dr. R. Sonnenschein: May I ask Dr. King whether he has had any experience with, or what his opinion is of, the use of iodized oil in these cases, in order to determine whether any marked change, such as thickening of the mucosa or polypoid formation, has occurred?

Dr. E. McGinnis, Chicago: I am in hearty accord with most everything that has been said. Dr. Shurly says that what he is interested in, in these nasal infections, is to get a shrinkage and drainage. There is one means of getting a shrinkage in the nasal mucosa that nobody has mentioned, namely, exercise. Some of these patients are too sick to get out of the house, and I am in the habit of having them go through a setting-up exercise, exercising the arms, dipping and so forth. I have found in my own case that if I go through these exercises they have the same effect as ephredine or any of the shrinking agencies of the nasal mucosa.

If the patient is able to get outdoors, I often start him out on a walk around one block, then around two blocks the next day. Sometimes I have them take two or three walks a day, and it is surprising how they will come back in two or three days and say, "The little bit of exercise that you advocated acts just the same as the nasal spray of ephredine solution."

I recently had a patient with nasal infection whom I had tried to get out for exercise, and he was prone to delay. He finally agreed to go down to Kentucky. There were plenty of hills in the neighborhood and he started walking up and down those hills. When he got back home he came into the office and he said, "One walk up a good hill is better than any intranasal treatment I have ever received."

Directory of Otolaryngologic and Ophthalmologic Societies *

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Robert C. Lynch, Maison Blanche Bldg., New Orleans, La.
Secretary: Dr. Gabriel Tucker, Bronchoscopic Clinic, University Hospital, Philadelphia.
Place: Detroit. Time: June 23-27, 1930.

American Bronchoscopic Society

President: Dr. Thomas E. Carmody, Metropolitan Bldg., Denver.
Secretary-Treasurer: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.

American Laryngological Association

President: Dr. Lewis A. Coffin, New York.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.

American Laryngological, Rhinological and Otological Society

President: Dr. Ross Hall Skillern, 1811 Spruce St., Philadelphia.
Secretary: Dr. R. L. Loughran, 33 E. 63d St., New York.
Place: Atlantic City, N. J. Time: May, 1930.

American Otological Society

President: S. MacCuen Smith, Philadelphia.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. Harris P. Mosher, 828 Beacon St., Boston.
President-Elect: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Atlantic City, N. J. Time: Oct. 21-25, 1929.

LOCAL

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

President: Dr. Harold S. Muckleston, 6422 Hollywood Blvd., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Place: California Hospital, assembly room. Time: 8 p. m., first Monday of each month.

The Colorado Oto-Laryngological Society

President: Dr. Frank L. Dennis, Ferguson Bldg., Colorado Springs.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg., Time: First Saturday of each month from October to May.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. Louis S. Greene, 1710 Rhode Island Ave. N. W., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month.

Atlanta Academy of Ophthalmology and Otolaryngology

President: Dr. H. F. McDuffie, 45 Edgewood Ave., Atlanta, Ga.
Secretary: Dr. Leon E. Brawner, 157 Forrest Ave..N. E., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: 8 p. m., last Thursday of each month.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. George B. Smith, Rome.
Secretary-Treasurer: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta.

Chicago Laryngological and Otological Society

President: Dr. Samuel Salinger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. Albert E. Bulson, 406 W. Berry St., Fort Wayne.
Secretary: Dr. Robert J. Masters, 601 Hume-Mansur Bldg., Indianapolis.
Place: French Lick Springs Hotel. Time: Dec. 12-13, 1929.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. Robert J. Masters, Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

New Orleans Ophthalmological and Otological Society

President: Dr. M. Earle Brown, Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. A. Wagner, Pere Marquette Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., third Thursday of each month from September to June.

Baltimore City Medical Society, Section on Otology and Laryngology

Chairman: Dr. Waitman F. Zinn, Medical Arts Bldg., Baltimore.
Secretary: Dr. Moses L. Breitstein, 1212 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

New England Otological and Laryngological Society

President: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary-Treasurer: Dr. Charles T. Porter, 520 Commonwealth Ave., Boston.

Detroit Oto-Laryngological Society

President: Dr. C. C. McClelland, David Whitney Bldg., Detroit.
Secretary-Treasurer: Dr. Don A. Cohoe, 13535 Woodward Ave., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

THE MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. C. N. Spratt, 900 Nicollet Ave., Minneapolis.
Secretary-Treasurer: Dr. J. H. Morse, 80 S. 7th St., Minneapolis.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. R. Forgrave, Logan Bldg., St. Joseph, Mo.
Secretary: Dr. A. E. Eubank, Argyle Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

ST. LOUIS EAR, NOSE AND THROAT CLUB

Acting President: Senior member of the executive committee: Dr. F. C. Simon, Arcade Bldg., St. Louis, Mo.
Secretary: Dr. M. L. Hanson, Murphy Bldg., East St. Louis, Ill.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Charles M. Swab, Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. J. C. Davis, 1615 Howard St., Omaha.
Place: Medical Arts Tea Room. Time: 5:15 and 7 p. m., third Wednesday of each month from October to May.

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Frederick J. Wort, 1080 Broad St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

NEW YORK ACADEMY OF MEDICINE, SECTION OF LARYNGOLOGY AND RHINOLOGY

Chairman: Dr. David H. Jones, 114 E. 54th St., New York.
Secretary: Dr. Edward Frankel, Jr., 152 W. 58th St., New York.
Place: 103d St. and 5th Ave. Time: 8:30 p. m., fourth Wednesday of each month from October to May.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OTOLOGY

Chairman: Dr. Mervin C. Myerson, 12 E. 86th St., New York.
Secretary: Dr. John M. Love, 240 West End Ave., New York.
Place: 103d St. and 5th Ave. Time: Second Friday of each month from October to May.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. John P. O'Keefe, 251 State St., Albany.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. A. Vincent Walker, 833 South Ave., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George M. Constans, Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

CINCINNATI OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Harris H. Vail, 24 E. 8th St., Cincinnati.
President-Elect: Dr. Edward King, Pearl Market Bank Bldg., Cincinnati.
Secretary-Treasurer: Dr. Donald Lyle, 19 W. 7th St., Cincinnati.
Place: University Club. Time: 6:45 p. m., second Tuesday of each month from October to May.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Myron Metzenbaum, Rose Bldg., Cleveland.
Secretary: Dr. A. D. Ruedemans, Euclid and E. 93d St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Arthur C. Jones, 105 N. 8th St., Boise, Idaho.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

PHILADELPHIA LARYNGOLOGICAL SOCIETY

President: Dr. Henry A. Laessle, 13th and Spruce Sts., Philadelphia.
Secretary: Dr. Herman B. Cohen, 13th and Spruce Sts., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLOGY AND LARYNGOLOGY

Chairman: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Clerk: Dr. Frederick M. Strouse, 13th and Spruce Sts., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. J. E. Raia, 272 Broadway, Providence.
Secretary: Dr. F. W. Dimmitt, Jr., 195 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William Thornwall Davis, 927 Farragut Sq., Washington, D. C.
Secretary: Dr. Fletcher D. Woodward, Box 162, University, Va.
Place: Miami, Fla. Time: Nov. 20-22, 1929.

THE MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. R. J. Warner, 706 Church St., Nashville.
Secretary-Treasurer: Dr. W. W. Wilkerson, Jr., Bennie-Dillon Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. D. L. Bettison, Medical Arts Bldg., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, Medical Arts Bldg., Dallas, Texas.
Recording Secretary: Dr. L. A. Nelson, 4105 Live Oak, Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. C. P. Schenck, Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, Fort Worth National Bank Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. John H. Foster, 1300 Walker Ave., Houston, Texas.
Secretary: Dr. Edward W. Griffey, Houston Clinic, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. F. H. Roseborough, 302 Moore Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

Utah Ophthalmological Society

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Ezra Thompson Bldg., Salt Lake City.

Virginia Society of Otolaryngology and Ophthalmology

President: Dr. H. B. Stone, 303 First St., Roanoke.
Secretary-Treasurer: Dr. Fletcher D. Woodward, Box 162, University.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Will Otto Bell, Medical-Dental Bldg., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, Medical-Dental Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. O. M. Rott, Paulsen Bldg., Spokane, Wash.
Secretary: Dr. C. A. Veasey, Jr., 421 Riverside Ave., Spokane, Wash.

The Milwaukee Oto-Ophthalmic Society

President: Dr. L. P. Allen, 145½ Main St., Oshkosh, Wis.
Secretary-Treasurer: Dr. Edward R. Ryan, 425 E. Water St., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

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